

# TRW PROPRIETARY INFORMATION

118496  
CHARACTERIZATION, RISK ASSESSMENT  
AND REMEDIAL ACTION PLAN  
FOR A PCB SPILL AT  
THE TRW SITE IN MINERVA, OHIO

Volume 2. Appendixes

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# TRW PROPRIETARY INFORMATION

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## APPENDIX A

Sample Number	Depth (in feet)	Concentration of PCB (in ppm*)	Date Collected	Date Reported
1a	6	14	7/7/81	7/24/81
1b	10	15	7/7/81	7/24/81
2	0.5	20	7/7/81	7/24/81
3a	0.5	348	7/7/81	7/24/81
3b	3	<2	7/15/81	7/24/81
4	0.5	1	7/15/81	7/24/81
5a	4.5	1,599	7/7/81	7/24/81
5b	8	38	7/15/81	7/24/81
6	0.5	320	7/7/81	7/24/81
7	0.5	734	7/7/81	7/24/81
8a	0.5	116	7/7/81	7/24/81
8b	2	91	7/15/81	7/24/81
9	0.5	<2	7/14/81	7/24/81
10	0.5	<2	7/14/81	7/24/81
11	0.5	<2	7/14/81	7/24/81
12	0.5	<2	7/14/81	7/24/81
13	0.5	8	7/14/81	7/24/81
14	1	<2	7/14/81	7/24/81
15	1	<2	7/14/81	7/24/81
16	0.5	7	7/14/81	7/24/81
17a	2.5	12	7/14/81	7/24/81
17b	0.5	1,073	7/14/81	7/24/81
18	0.5	9	7/14/81	7/24/81
19	0.5	7	7/14/81	7/24/81
20	0.5	36	7/14/81	7/24/81
21	0.8	23	7/14/81	7/24/81
22	0.5	1,060	7/14/81	7/24/81
23	Raw creek effluent	0.7 ppb	7/14/81	7/24/81
24	Effluent	1 ppb	7/14/81	7/24/81
25	Surface	399	7/14/81	7/24/81
26	0.8	293	7/15/81	7/24/81
27	0.8	186	7/15/81	7/24/81
28	0.8	46	7/15/81	7/24/81
29a	6.0	426	7/15/81	7/24/81
29b	10.0	59	7/15/81	7/24/81
30	Water	<1 ppb	7/22/81	7/29/81
31	Water	<1 ppb	7/22/81	7/29/81
32	Sediment	<2	7/22/81	7/29/81
33	Water	<1 ppb	7/22/81	7/29/81
34	Soil	4	7/22/81	7/29/81
35	Water	<1 ppb	7/22/81	7/29/81

\*Water samples are reported in ppb.

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Sample Number	Depth (in feet)	Concentration of PCB (in ppm*)	Date Collected	Date Reported
36	Water	<1 ppb	7/22/81	7/29/81
37	Sediment	<2	7/22/81	7/29/81
38	0.5	2	7/28/81	8/7/81
39	0.5	2	7/28/81	8/7/81
40	0.5	<1	7/28/81	8/7/81
41	0.5	1	7/28/81	8/7/81
42	0.5	5	7/28/81	8/7/81
43	0.5	2	7/28/81	8/7/81
44	0.5	428	7/28/81	8/7/81
45	0.5	1	7/28/81	8/7/81
46	0.5	136	7/28/81	8/7/81
47	0.5	<1	7/28/81	8/7/81
48	Water	8 ppb	7/28/81	8/7/81
49	Slab	207	7/28/81	8/7/81
50	0.5	60	7/28/81	8/7/81
51	0.5 (Sed)	3	7/28/81	8/7/81
52	0.5 (Sed)	21	7/28/81	8/7/81
53	Plant effluent	1 ppb	7/28/81	8/7/81
55	Effluent	6 ppb	7/28/81	8/7/81
56	Water	2 ppb	7/28/81	8/7/81
57	0.5 (sed)	27	7/28/81	8/7/81
58a	0.5	108	8/5/81	8/10/81
58b	2.0	95	8/5/81	8/10/81
58c	4.0	1	8/5/81	8/10/81
59a	Surface	249	8/5/81	8/10/81
59b	2.0	1	8/5/81	8/10/81
59c	4.0	3	8/5/81	8/10/81
60a	Surface	62	8/5/81	8/10/81
60b	2.0	<1	8/5/81	8/10/81
60c	4.0	<1	8/5/81	8/10/81
61a	Surface	27	8/5/81	8/10/81
61b	2.0	<1	8/5/81	8/10/81
61c	4.0	<1	8/5/81	8/10/81
62a	Surface	1,613	8/5/81	8/10/81
62b	2.0	5	8/5/81	8/10/81
62c	4.0	<1	8/5/81	8/10/81
63a	Surface	176	8/5/81	8/10/81
63b	2.0	3	8/5/81	8/10/81
63c	4.0	1	8/5/81	8/10/81

\*Water samples are reported in ppb

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
64a	Surface	331	8/5/81	8/10/81
64b	2.0	1	8/5/81	8/10/81
64c	4.0	2	8/5/81	8/10/81
65	Surface	204	8/5/81	8/10/81
	2.0	3	8/5/81	8/10/81
66	Surface	1,252	8/5/81	8/10/81
	2.0	7	8/5/81	8/10/81
67	Surface	178	8/5/81	8/10/81
	2.0	1	8/5/81	8/10/81
	4.0	<1	8/5/81	8/10/81
68	Surface	17	8/5/81	8/10/81
	2.0	<1	8/5/81	8/10/81
	4.0	<1	8/5/81	8/10/81
69	Surface	15	8/5/81	8/10/81
	2.0	<1	8/5/81	8/10/81
	4.0	<1	8/5/81	8/10/81
70	Surface	29	8/5/81	8/10/81
	2.0	<1	8/5/81	8/10/81
	4.0	<1	8/5/81	8/10/81
71	Surface	1,168	8/5/81	8/10/81
	3.0	722	8/5/81	8/10/81
72	Scrape inside building	7	8/5/81	8/10/81
73				
74	New pad area (Underneath)	3,768	8/5/81	8/10/81
75	7.0 (Sediment)	17	8/5/81	8/10/81
76				
77	See map for location (SW corner of plant)			
78				
79a	2.5	<1		8/19/81
79b	4.5	<1		8/19/81
80a	2.0	<1		8/19/81
80b	5.0	<1		8/17/81
81	Surface	4		8/19/81
82	Surface	11		8/19/81
83	Surface	4		8/19/81
84	Surface	10		8/19/81
85	Surface	6		8/19/81
86	Surface	<1		8/19/81
87	Surface	<1		8/19/81
88	Surface	<1		8/19/81
89	Surface	1		8/19/81

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Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
90a	Surface	660		8/19/81
90b	2.0	73		8/19/81
90c	4.0	<1		8/19/81
91a	Surface	1,057		8/19/81
91b	2.0	190		8/19/81
91c	4.0	<1		8/19/81
92a	Surface	616		8/19/81
92b	2.0	<1		8/19/81
93a	Surface	734		8/19/81
93b	2.0	71		8/19/81
93c	7.0	5		8/19/81
94a	Surface	1,388		8/19/81
94b	2.0	50		8/19/81
94c	4.0	37		8/19/81
95a	Surface	239		8/19/81
95b	2.0	29		8/19/81
95c	4.0	<1		8/19/81
96a	Surface	445		8/19/81
96b	2.0	301		8/19/81
96c	4.0	18		8/19/81
97a	Surface	566		8/19/81
97b	2.0	12		8/19/81
97c	4.0	4		8/19/81
98a	Surface	1,298		8/19/81
98b	2.0	9		8/19/81
99a	Surface	338		8/19/81
99b	2.0	15		8/19/81
99c	4.0	1		8/19/81
100a	Surface	240		8/19/81
100b	2.0	5		8/19/81
101a	Surface	959		8/19/81
101b	2.0	4		8/19/81
102	See map for location (SW corner of plant)			
103a	Surface	201		8/19/81
103b	2.0	8		8/19/81
104a	Surface	1,095		8/19/81
104b	2.0	9		8/19/81
105a	Surface	47		8/19/81
105b	2.0	2		8/19/81
106a	Surface	150		8/19/81
106b	2.0	130		8/19/81
107	South surface	81		8/19/81
108	North surface	11		8/19/81
109	?	3		8/19/81
110	Surface	3		8/19/81

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Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
111	Surface	108		8/19/81
112	Surface	25		8/19/81
113	Surface	149		8/19/81
114	Surface	9		8/19/81
115a-117a	Surface (composite)	90		8/21/81
115b-117b	2.0 (composite)	<1		8/21/81
118a-120a	Surface (composite)	426		8/21/81
121a-123a	Surface (composite)	71		8/21/81
122b-124b	2.0 (composite)	<1		8/21/81
124a-126a	Surface (composite)	72		8/21/81
127a-129a	Surface (composite)	36		8/21/81
130a-132a	Surface (composite)	3		8/21/82
133a-135a	Surface (composite)	3		8/21/81
136a-138a	Surface (composite)	4		8/21/81
139a-141a	Surface (composite)	3		8/21/81
142a-144a	Surface (composite)	3		8/21/81
145a-147a	Surface (composite)	1		8/21/81
148	Surface	<1		9/11/82
149	Surface	10		9/11/82
150	Surface	2		9/11/82
150b-151b	2.0 (composite)	<1		9/11/82
151	Surface	13		9/11/82
152a	Surface	4		9/11/82
152b-153b	2.0 (composite)	<1		9/11/82
153	Surface	20		9/11/82
154b-156b	2.0 (composite)	<1		9/11/82
154 <sup>a</sup> -156 <sup>a</sup>	Surface (composite)	4		9/11/82
157 <sup>a</sup> -159 <sup>a</sup>	Surface composite	1		9/11/82
P1a	0.5	170 (Aro 1254) *		9/9/81
		32 (Aro 1242)		9/9/81
P1b	1.0	32 (Aro 1254)		9/9/81
		5 (Aro 1242)		9/9/81
P2a	0.5	332 (Aro 1254)		9/9/81
		130 (Aro 1242)		9/9/81
P2b	1.0	42 (Aro 1254)		9/9/81
		9 (Aro 1242)		9/9/81
P3a	0.5	180 (Aro 1252)		9/9/81
		58 (Aro 1242)		9/9/81
P3b	1.0	<1 (Aro 1254)		9/9/81
		<1 (Aro 1242)		9/9/81
P4a	0.5	433 (Aro 1254)		9/9/81
		470 (Aro 1242)		9/9/81
P4b	1.0	600 (Aro 1254)		9/9/81
		208 (Aro 1242)		9/9/81

\*Aro=Aroclor

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Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
SP1 (SL1)	Surface	1,732 (Aro 1254)	9/22/81	10/16/81
	Sediment	1,556 (Aro 1254)	9/22/81	10/16/81
		290 (Aro 1242)	9/22/81	10/16/81
		215 (Aro 1242)	9/22/81	10/16/81
SP2 (SL2)	Surface	156 (Aro 1254)	9/22/81	10/16/81
	Sediment	214 (Aro 1242)	9/22/81	10/16/81
SP3 (SL3)	Surface	441 (Aro 1254)	9/22/81	10/16/81
	Sediment	382 (Aro 1242)	9/22/81	10/16/81
SP4 (SL4)	Surface	197 (Aro 1254)	9/22/81	10/16/81
	Sediment	183 (Aro 1242)	9/22/81	10/16/81
SP5 (SL5)	Surface	516 (Aro 1254)	9/22/81	10/16/81
	Sediment	64 (Aro 1242)	9/22/81	10/16/81
SP6 (SL6)	Surface	597 (Aro 1254)	9/22/81	10/16/81
	Sediment	176 (Aro 1242)	9/22/81	10/16/81
SP7 (SL7)	Surface	17,20 (Aro 1254)	9/22/81	10/16/81
	Sediment	8,4 (Aro 1242)	9/22/81	10/16/81
SP8 (SL8)	Surface	609 (Aro 1254)	9/22/81	10/16/81
	Sediment	67 (Aro 1242)	9/22/81	10/16/81
SP9 (SL9)	Surface	128 (Aro 1254)	9/22/81	10/16/81
	Sediment	26 (Aro 1242)	9/22/81	10/16/81
SP10 (SL10)	Sediment	1,123 (Aro 1254)	9/22/81	10/16/81
	Surface	311 (Aro 1242)	9/22/81	10/16/81
SP11 (SL11)	Surface	570 (Aro 1254)	9/22/81	10/16/81
	Sediment	96 (Aro 1242)	9/22/81	10/16/81
SP12 (SL12)	Surface	320 (Aro 1254)	9/22/81	10/16/81
	Sediment	138 (Aro 1242)	9/22/81	10/16/81
SP13 (SL13)	Surface	1,523 (Aro 1254)	9/22/81	10/16/81
	Sediment	258 (Aro 1242)	9/22/81	10/16/81
SP14 (SL14)	Surface	630 (Aro 1254)	9/22/81	10/16/81
	Sediment	481 (Aro 1254)	9/22/81	10/16/81
		150 (Aro 1242)	9/22/81	10/16/81
		137 (Aro 1242)	9/22/81	10/16/81
(G1) 3001	0.5	15		1/30/82
(G2) 3002	1.5	77		1/30/82
(G3) 3003	2.5	307		1/30/82
(G4) 3004	3.5	7		1/30/82
(G5) 3005	4.5	<5		1/30/82
(G6) 3006	5.5	474		1/30/82
(G7) 3007	0.5	1,024		1/30/82
(G8) 3008	1.5	13		1/30/82
(G9) 3009	2.5	<5		1/30/82
(G10) 3010	3.5	7		1/30/82
(G11) 3011	4.5	<5		1/30/82
(G12) 3012	0.5	<1		1/30/82
(G13) 3013	1.5	<5		1/30/82
(G14) 3014	2.5	31		1/30/82



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Sample Number	Depth (in feet)	Concentration of PCB (in ppm*)	Date Collected	Date Reported
(G15) 3015	3.5	8		1/30/82
(G16) 3016	0.5	<5		1/30/82
(G17) 3017	1.5	15		1/30/82
(G18) 3018	2.5	<5		1/30/82
(G19) 3019	0.5	10		1/30/82
(G20) 3020)	1.5	<5		1/30/82
FP1	Top 0.5	<1	2/25/82	3/9/82
FP2	Top 0.5	<1	2/25/82	3/9/82
FP3	Top 0.5	<1	2/25/82	3/9/82
FP4	Top 0.5	<1	2/25/82	3/9/82
FP5	Top 0.5	22,000	2/25/82	3/9/82
4-28-1 (1F1)	0.25	90	4/28/82	5/11/82
4-28-2 (1F2)	0.25	32,700	4/28/82	5/11/82
4-28-3 (1F3)	0.25	3,780	4/28/82	5/11/82
4-28-4 (1F4)	0.25	20	4/28/82	5/11/82
4-28-5 (1F5)	0.25	10	4/28/82	5/11/82
4-28-6 (1F6)	0.25	1,950	4/28/82	5/11/82
4-28-7 (1F7)	0.25	3	4/28/82	5/11/82
4-28-8 (1F8)	0.25	1	4/28/82	5/11/82
4-28-9 (1F9)	0.25	1	4/28/82	5/11/82
4-28-10 (1F10)	0.25	<1	4/28/82	5/11/82
4-28-11 (1F11)	0.25	<1	4/28/82	5/11/82
WE	Water	<1 ppb	4/28/82	5/11/82
EE	Water	<1 ppb	4/28/82	4/11/82
5-25-1 (2F1)	0.25	<1	4/25/82	6/7/82
5-25-2 (2F2)	0.25	96	5/25/82	6/7/82
5-25-3 (2F3)	0.25	560	5/25/82	6/7/82
5-25-4 (2F4)	0.25	2	5/25/82	6/7/82
5-25-5 (2F5)	0.25	<1	5/25/82	6/7/82
5-25-6 (2F6)	0.25	100	4/25/82	6/7/82
5-25-7 (2F7)	0.25	4,200	5/25/82	6/7/82
5-25-8 (2F8)	0.25	4	5/25/82	6/7/82
5-25-9 (2F9)	0.25	55	5/25/82	6/7/82
5-25-10 (2F10)	0.25	<1	5/25/82	6/7/82
5-25-11 (2F11)	0.25	1	5/25/82	6/7/82
5-25-12 (2F12)	0.25	10	5/25/82	6/7/82
5-25-13 (2F13)	0.25	2	5/25/82	6/7/82
5-25-14 (2F14)	0.25	<1	5/25/82	6/7/82
5-25-15 (2F15)	0.25	<1	5/25/82	6/7/82
5-25-16 (2F16)	0.25	<1	5/25/82	6/7/82
5-25-17 (2F17)	0.25	13	5/25/82	6/7/82

\*Water samples are reported in ppb

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Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
R 1-0	Surface	130		
R 2-0	Surface	80		
R 3-0	Surface	5		
R 4-0	Surface	7		
R 5-0	Surface	8		
R 6-0	Surface	340		
R 7-0	Surface	740		
R 8-0	Surface	10		
R 9-0	Surface	500		
5-18-1A(R 1A-0)	Surface	98		
5-19-1A-1(R 1A-1)	1	5		
5-18-2A(R 2A-0)	Surface	118		
5-19-2A-1(R 2A-1)	1	1		
5-18-3A(R 3A-0)	Surface	14		
5-19-3A-1(R 3A-1)	1	29		
5-19-3A-2(R 3A-2)	2	20		
5-18-4A(R 4A-0)	Surface	152		
5-19-4A-1(R 4A-1)	1	<1		
5-18-5A(R 5A-0)	Surface	26		
5-18-5A-1(R 5A-1)	1	220		
5-18-5A-2(R 5A-2)	2	<1		
5-18-6A-1(R 5-1)	1	<1		
5-18-7A-1(R 3-1)	1	<1		
5-19-20A-1(R 7-1)	1	580		
5-19-20A-2(R 7-2)	2	<1		
5-19-21A-1(R 1-1)	1	<1		

## South Pond Berm

501	Surface	2
502	1.5	<1
503	Surface	2
504	Surface	3
505	1.5	2
506	Surface	98
507	2.5	170
508	1.0	18
509	3.5	69
510	Surface	12

7/9/82  
M. J. ...

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Rubble Pile

511a	Surface	5		
511b	1.5	23		
512	Surface	9		
513a	Surface	3		
513b	1.5	<1		
514	Surface	2		
515	Surface	17		
516	Surface	12		
517	Surface	6		
518a	Surface	1		
518b	1.5	3		
519	Surface	614		
520a	Surface	139		
520b	1.5	28		
521	Surface	500		
522	Surface	141		
523	Surface	136		
524	Surface	955		
525a	Surface	438		
525b	1.5	5		
526a	Surface	320		
526b	1.5	330		
527	Surface	580		
528a	Surface	21		
528b	1.5	<1		
529	Surface	110		
530a	Surface	10		
530b	1.5	<1		
531	Surface	<1		
532	Surface	<1		
533	Surface	136		
534	Surface	4,960		

## Sediment Samples Upstream from West Lake

535	Surface	<1		
536	Surface	<1		
537	Surface	<1		
538	Surface	<1		
539	Surface	<1		

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
------------------	--------------------	----------------------------------	-------------------	------------------

## West Lake Sediments

540	Surface	2		
541	Surface	2		
542	Surface	6		
543	Surface	<1		
544	Surface	121		

## Downstream of Swale

545	1.0	<1		
546	3.5	<1		
547	Surface <sup>b</sup>	<1		
548	1.0	<1		
549	1.0	<1		
550	3.0	<1		
551	4.5	<1		
552	Surface	17		
553	Surface	<1		
554	Surface	<1		
555	4.5	<1		
556	1.0	<1		
557	Surface	<1		
558	Surface	<1		
559	Surface	<1		
560	1.5	<1		
561	Surface	<1		
562	1.0	4		
563	Surface	<1		
564	Surface	<1		
565	1.0	<1		

## Well Core Analysis (566-578)

Well No.				
10	3	<1		
	6	<1		
11	3	<1		
	6	<1		
	9	<1		
	12	<1		
13	3	<1		
	6	<1		

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
<u>Wax Ditch</u>				
579	Surface	Not collected		
580	2.0	Not collected		
581	Surface	Not collected		
582	4.0	Not collected		
583	Surface	Not collected		
584	2.0	Not collected		
585	8.5	Not collected		
586	1.0	Not collected		
587	4.0	Not collected		
588	Surface	Not collected		
589	4.0	Not collected		
590	2.5	Not collected		
591	2.5	Not collected		
592	Surface	Not collected		
593	3.0	Not collected		
594	1.0	Not collected		
595	9.5	Not collected		
596	5.0	Not collected		
597	5.0	Not collected		
598	1.5	Not collected		
599	Surface	Not collected		
600	1.5	Not collected		
601	8.0	Not collected		
602	4.5	Not collected		
603	1.0	Not collected		
604	Surface	Not collected		
605	1.5	Not collected		
606	5.0	Not collected		
607	Surface	Not collected		
608	6.5	Not collected		
609	1.5	Not collected		
610	4.5	134		
611	(Surface--	5,720)		
612	1.0	4,370		
613	4.0	Not collected		
614	1.0	Not collected		
615	3.0	5,140		
616	6.5	Not collected		
617	Surface	Not collected		
618	4.5	102		

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Low Area West of South Pond

619	Surface	<1		
620	Surface	<1		
621	Surface	<1		
622	1.5	<1		
623	Surface	<1		
624	Surface	<1		
625	Surface	<1		
626	Surface	<1		
627	Surface	<1		
628	1.5	<1		
629	Surface	<1		
630	1.5	<1		

## North Property Line of TRW

631	Surface	<1		
632	Surface	Not collected		
633	Surface	<1		
634	Surface	Not collected		
635	Surface	<1		
636	Surface	Not collected		
637	Surface	<1		
638	Surface	Not collected		
639	Surface	<1		
640	Surface	Not collected		
641	Surface	<1		
642	Surface	Not collected		
643	Surface	<1		
644	Surface	Not collected		
645	Surface	<1		
646	Surface	Not collected		
647	Surface	<1		
648	Surface	Not collected		
649	Surface	<1		
650	Surface	Not collected		

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Section B of the Swale

651	1.5	20
652	6.0	5
653	2.5	147
654	9.0	<1
655	2.5	1
656	1.0	103
657	1.0	247
658	5.0	131
659	5.5	5
660	4.0	45
661	1.0	27
662	4.5	167
663	4.0	3
664	1.0	9
665	5.5	110
666	2.5	3
667	9.0	188
668	2.0	<1
669	2.5	<1
670	6.0	<1
671	5.5	1
672	2.0	<1
673	10.0	<1
674	6.0	<1
675	1.0	<1
676	5.0	54
677	6.0	62
678	4.0	8
679	7.0	2
680	4.0	3
681	8.0	2
682	1.0	2
683	9.0	<1
684	10.0	17
685	4.0	7
686	1.0	15
687	1.0	91
688	1.5	<1

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Corner Section of the Swale

689	9.5	3
690	13.0	<1
691	6.5	<1
692	12.0	<1
693	11.5	19
694	7.5	2
695	13.5	10
696	14.5	6
697	11.0	28
698	9.5	11
699	10.0	52
700	13.5	<1
701	9.0	5
702	15.0	<1
703	11.5	<1
704	7.5	<1
705	7.5	6>
706	12.5	19
707	9.0	835
708	12.0	77
709	6.0	151
710	15.0	3
711	9.5	2
712	6.5	2

## Core Samples in South Pond

713a	Surface	67
b	1.5	Clay*
c	2.5	
d	4.0	
714a	1.0	Not collected
b	1.5	
c	2.5	
d	4.0	

\*Deeper samples could not be collected because clay was encountered.  
Clay is at least 1 foot thick.



# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Core Samples in South Pond (continued)

715a	Surface	24		
b	1.5	Clay		
c	2.5			
d	4.0			
716a	Surface	430		
b	1.0	32		
c	2.5	Clay		
d	4.0			
717a	Surface	290		
b	1.0	19		
c	2.0	23		
d	4.0	Clay		
718a	Surface	28		
b	1.5	1.5		
c	2.5	Clay		
d	4.0			
719a	1.0	Not collected		
b	1.5			
c	2.5			
d	4.0			
720a	1.0	Not collected		
b	1.5			
c	2.5			
d	4.0			

## Fence Line Samples Near Rubble Pile

721	Surface	377
722	Surface	56
723	Surface	226
724	Surface	13
725	Surface	<1
726	Surface	<1
727	Surface	<1
728	Surface	<1
729	Surface	<1
730	Surface	<1
731	Surface	<1

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Grid Sample for Fry Property

732	Surface	<1		
733	2.0	<1		
734	1.0	1		
735	Surface	<1		
736	Surface	279		
737	Surface	1		
738	1.0	<1		
739	Surface	<1		
740	1.0	<1		
741	1.0	<1		
742	1.0	17		
743	1.0	<1		
744	Surface	1		
745	Surface	<1		
746	Surface	1		
747	Surface	<1		
748	Surface	<1		
749	1.0	<1		
750	Surface	<1		
751	Surface	<1		
752	Surface	<1		
753	2.0	<1		
754	Surface	<1		
755	Surface	<1		
756	Surface	<1		
757	Surface	<1		
758	Surface	171		
759	1.0	7		
760	2.0	<1		
761	1.0	<1		
762	Surface	<1		
763	Surface	<1		
764	Surface	20		
765	1.0	<1		
766	Surface	<1		
767	Surface	10		
768	1.0	<1		
769	1.0	<1		
770	Surface	<1		
771	Surface	<1		
772	Surface	<1		
773	Surface	<1		
774	Surface	<1		
775	1.0	<1		
776	2.0	<1		
777	1.0	<1		

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Rubble Pile Core

778a	1.5	63		
b	3.0	30		
c	6.0	<1		
779a	1.5	217		
b	3.0	Not collected		
c	6.0	Not collected		
780a	1.5	79		
b	3.0	405		
c	6.0	15		
d	9.0	385		
781a	1.5	244		
b	3.0	43		
c	6.0	1		
782a	1.5	11		
b	3.0	50		
c	6.0	<1		
783a	1.5	3		
b	3.0	3		
c	6.0	<1		
d	9.0	<1		
784a	1.5	990		
b	3.0	3		
c	6.0	<1		
785a	1.5	23		
b	3.0	Not collected		
c	6.0	Not collected		
786a	1.5	34		
b	3.0	6		
c	6.0	<1		
d	9.0	<1		
787a	1.5	628		
b	3.0	39		
c	6.0	103		
788a	1.5	<1		
b	3.0	<1		
c	6.0	<1		
789a	1.5	9		
b	3.0	<1		
c	6.0	<1		
d	9.0	<1		
790a	1.5	3		
b	3.0	<1		
c	6.0	<1		

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
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## Rubble Pile Core (continued)

791a	1.5	8
b	3.0	<1
c	6.0	<1
792a	1.5	231
b	3.0	<1
c	6.0	<1
d	9.0	<1

## East Fence by Parking Lot

793	Surface	<1
794	Surface	<1
795	Surface	<1
796	Surface	<1
797	Surface	<1
798	Surface	<1
799	Surface	<1
800	Surface	<1

## Front Property (west side of plant)

801	Surface	<1
802	Surface	<1
803	Surface	<1
804	Surface	1
805	Surface	<1
806	Surface	<1
807	Surface	<1
808	Surface	<1

## Storage-Shed Area

809	Surface	8
810	Surface	9
811	Surface	12
812	Surface	3
813	Surface	Not collected

## Parking Lot Runoff

814 (PL1)	Composite	1
815 (PL2)	Surface	<1
816 (PL3)	Surface	1
817 (R1)	Surface	3
818 (R2)	Surface	1

# TRW PROPRIETARY INFORMATION

Sample Number	Depth (in feet)	Concentration of PCB (in ppm)	Date Collected	Date Reported
<u>Decachlor Analysis</u>			3/30/83	5/02/83
819	1.5	3800 (<1) <sup>a</sup>		
820	4.5	500 (<1) <sup>a</sup>		
821	1.5	460 (<1) <sup>a</sup>		
822	4.5	85 (<1) <sup>a</sup>		
823	Surface	1440 (<1) <sup>a</sup>		
824	Surface	1600 (<1) <sup>a</sup>		

<sup>a</sup>Concentration of decachlor in sample in parenthesis

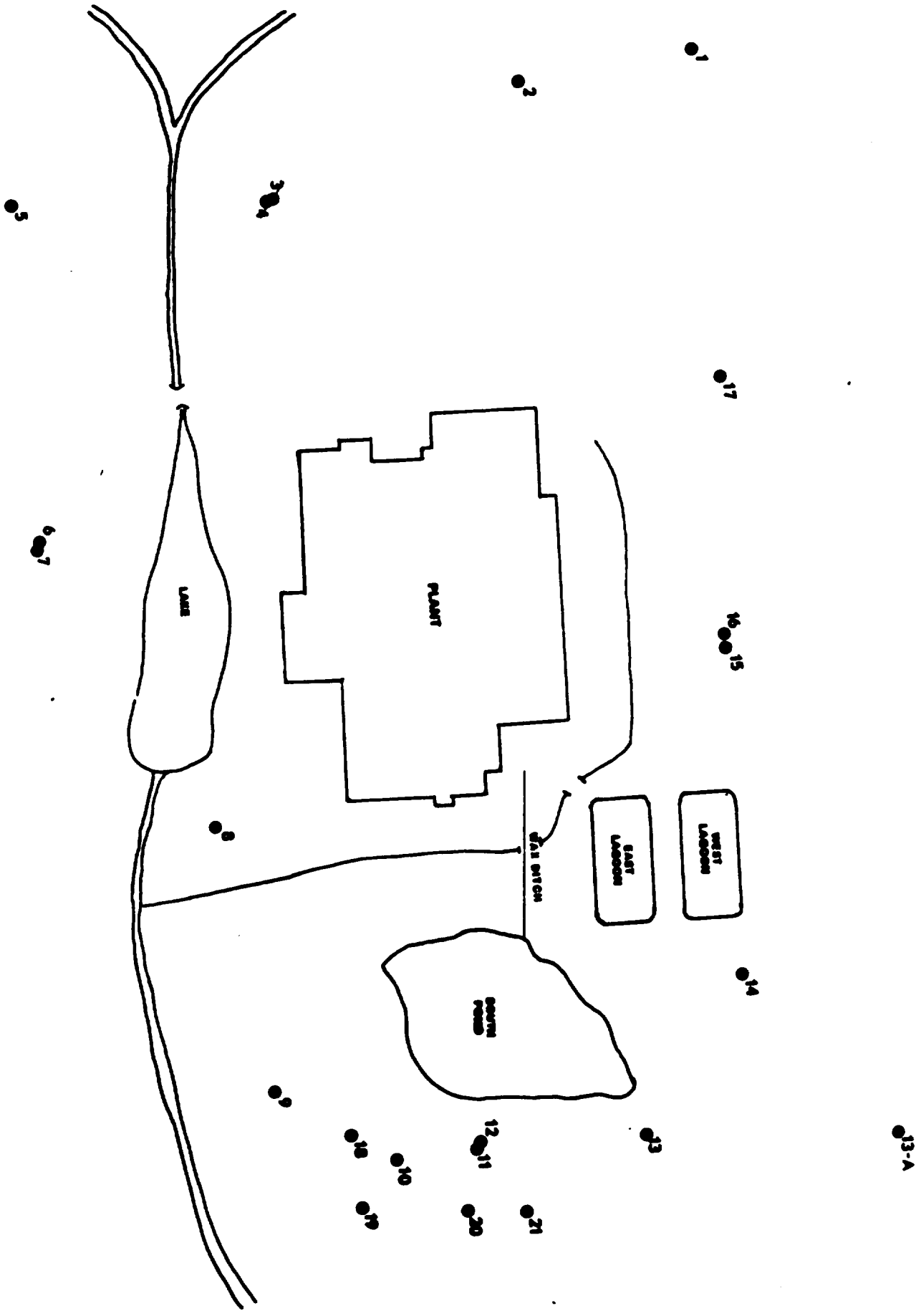


Figure A.1

# TRW PROPRIETARY INFORMATION

## APPENDIX B

### GROUNDWATER COMPUTER MODEL

The movement of PCB contamination in groundwater was modeled with the aid of a computer. Initially, a linear (one-dimensional) model was employed to provide a preliminary profile of potential groundwater contaminant plumes (Ogata and Banks 1961). Then potential plumes were modeled using a more sophisticated two-dimensional approach. The two-dimensional models were developed in two stages. First, groundwater levels from the monitoring wells were put into a program by Yeh and Ward (1980) to determine the pattern of groundwater flow in the region. Soil parameters necessary for the model were obtained from a soil survey of Stark County (USDA 1971). The output from this model was then combined with source strength parameters derived for the various point sources on the site and input into a second model to determine the dispersion of contamination within the groundwater as a function of time. The second model is run on a program by Yeh and Ward (1981). Both programs are based on the principals of finite difference techniques. Results are presented below.

Results of the two-dimensional model of contaminant movement from a line source at intervals of 25 years, 100 years, 300 years, and 500 years is presented in Figures B.1, B.2, B.3, and B.4, respectively. The x-axis represents distance in feet downgradient from the source. The y-axis represents distance in feet from the center-line of the source. Each isopleth (line of constant

FIGURE B.1

# HORIZONTAL MODEL

CONTAMINANT DISTRIBUTION AT 25 YEARS

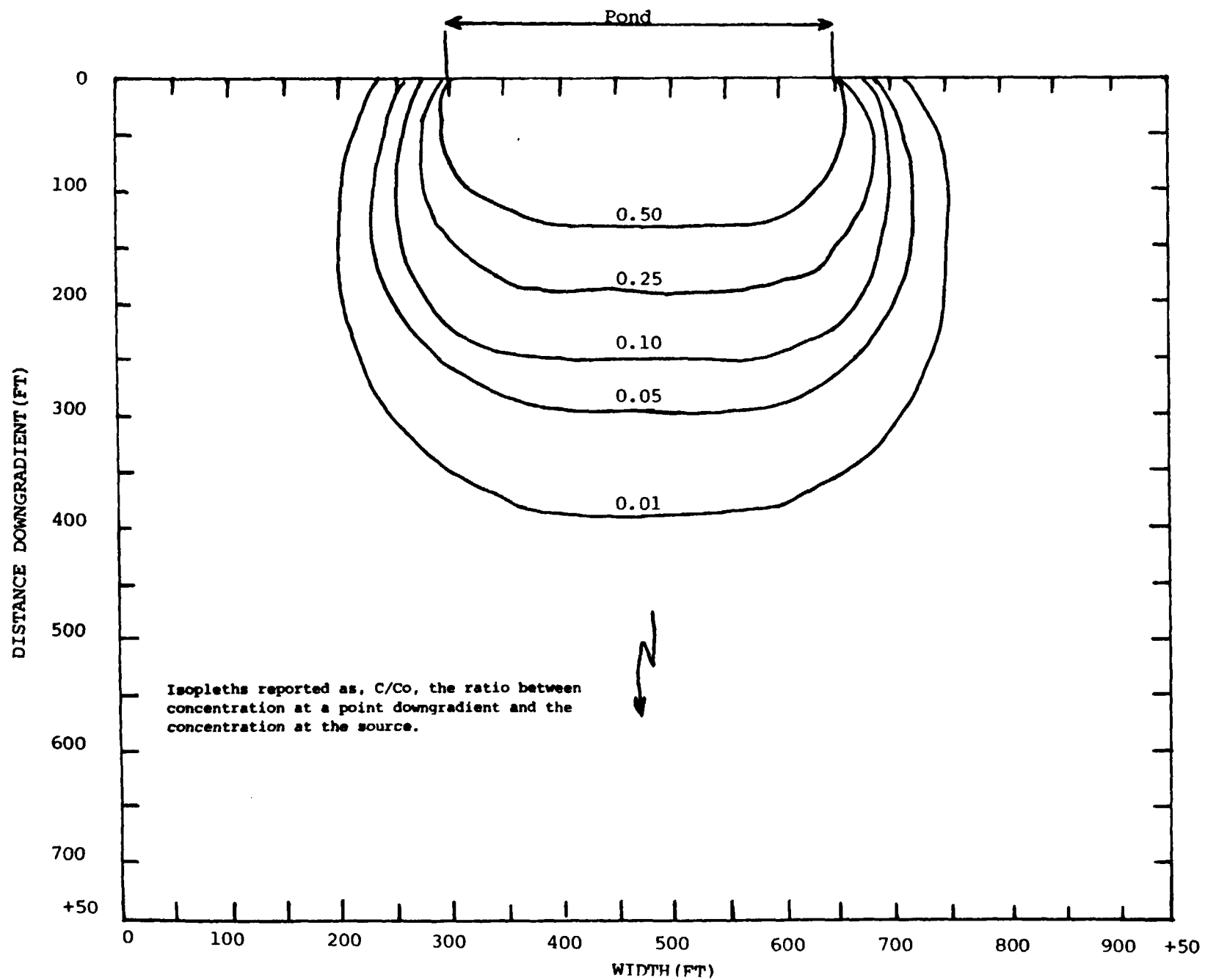
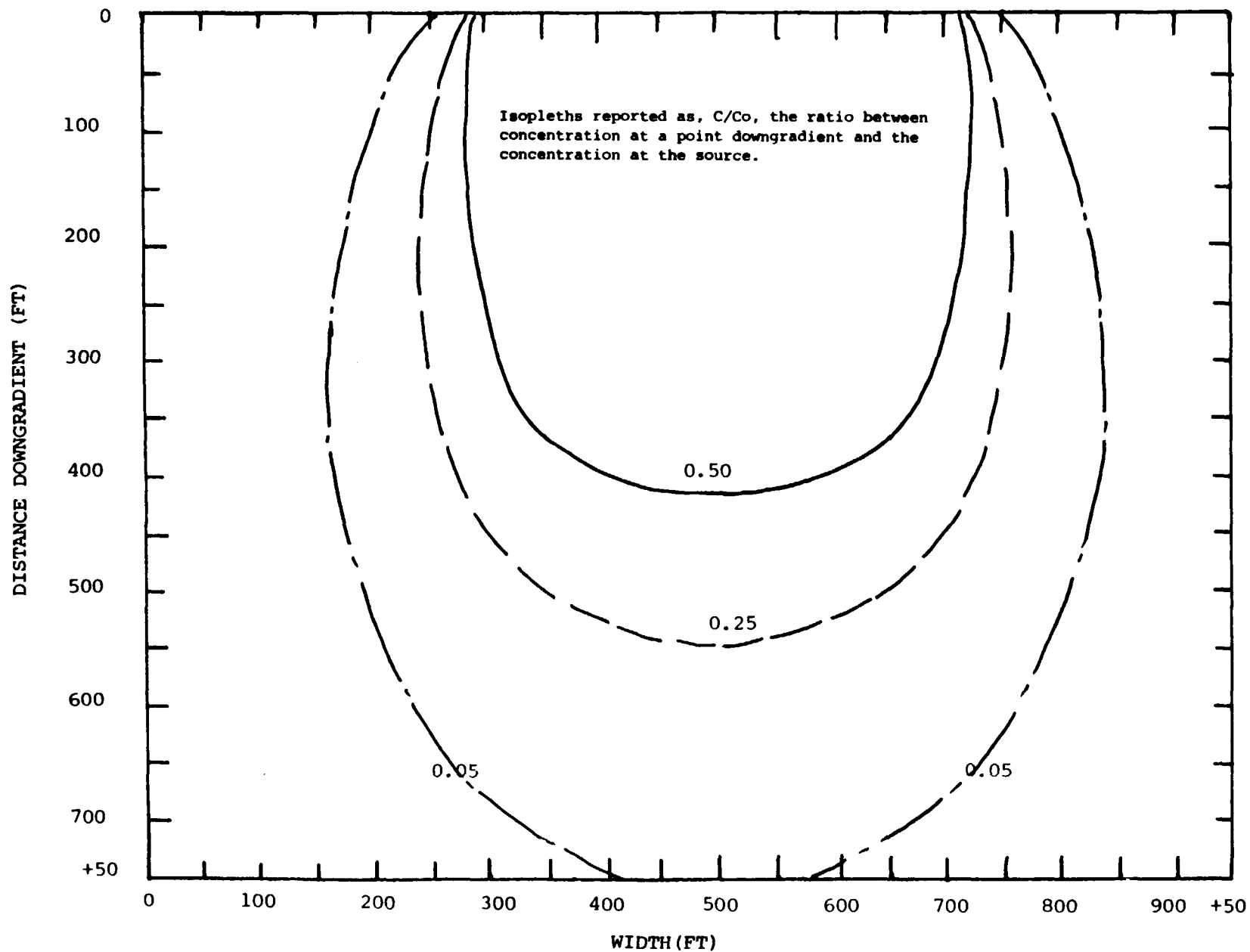




FIGURE B.2

HORIZONTAL MODEL: NO REMOVAL

CONCENTRATION DISTRIBUTION AT 100 YEARS



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FIGURE B.3

HORIZONTAL MODEL: NO REMOVAL

CONCENTRATION DISTRIBUTION AT 300 YEARS

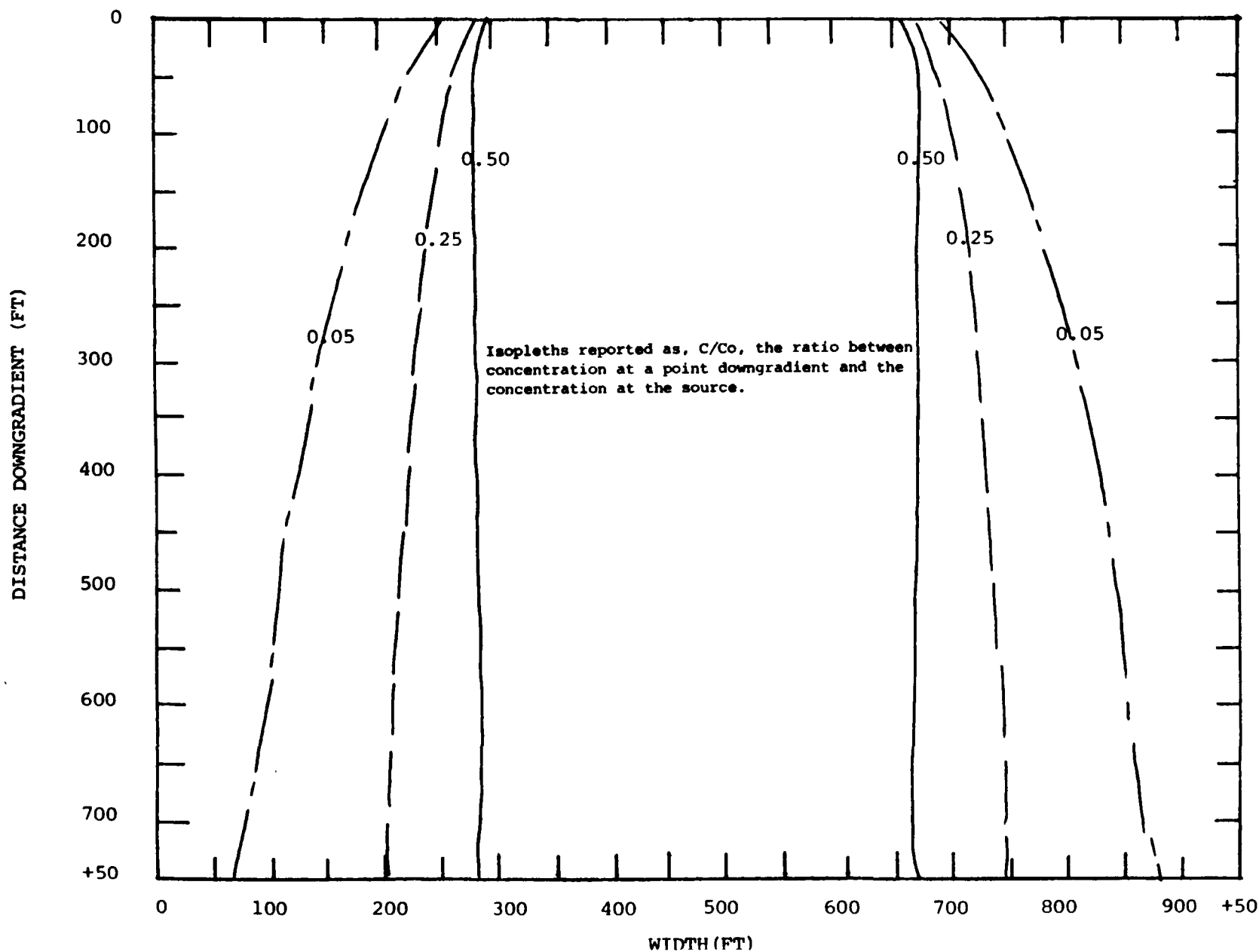
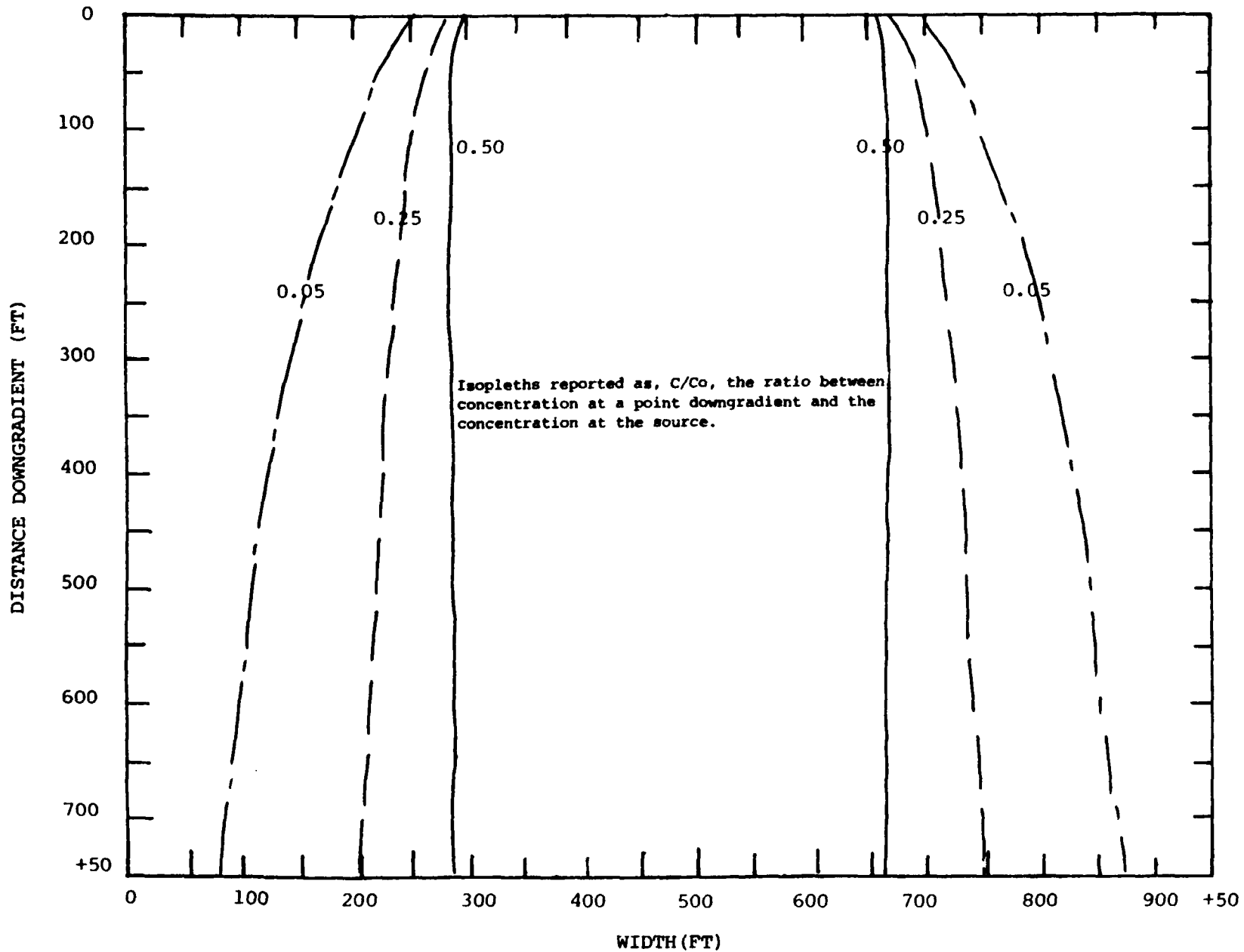


FIGURE B.4

HORIZONTAL MODEL: NO REMOVAL

CONCENTRATION DISTRIBUTION AT 500 YEARS



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# TRW PROPRIETARY INFORMATION

concentration) represents,  $C/C_0$ , where the concentration,  $C$ , at each point in space and time is represented as a fraction of the source-strength,  $C_0$ . For example, the concentration of a contaminant plume on the center line of the plume 125 feet from the source is one-half the concentration at the source after 25 years (Figure B.1). (Since projections are symmetric with respect to the center line of the plume, only the right half of each isopleth is presented on projections after 25 years.)

The influx rate,  $Q$ , affects the profile of a contaminant plume principally by affecting the mixing depth of the plume. (A mixing depth is the effective vertical extent of contamination in an aquifer.) To assess this dependence, a vertical plume model was run to profile the vertical extent of a contaminant plume emanating from South Pond after 25 years. The worst case (high  $Q$ ) plume is depicted in Figure B.5, and the low flow probable case is depicted in Figure B.6. The most obvious difference between these two cases is that the entire aquifer (to a depth of 100 feet) will become contaminated in the high flow case, while in the low flow case, the contaminant plume tends to remain at the surface of the aquifer. (Note: The horizontal model results apply most directly to the low flow case.)

FIGURE B.5

VERTICAL MODEL: HIGH FLOW

CONTAMINANT DISTRIBUTION AT 25 YEARS

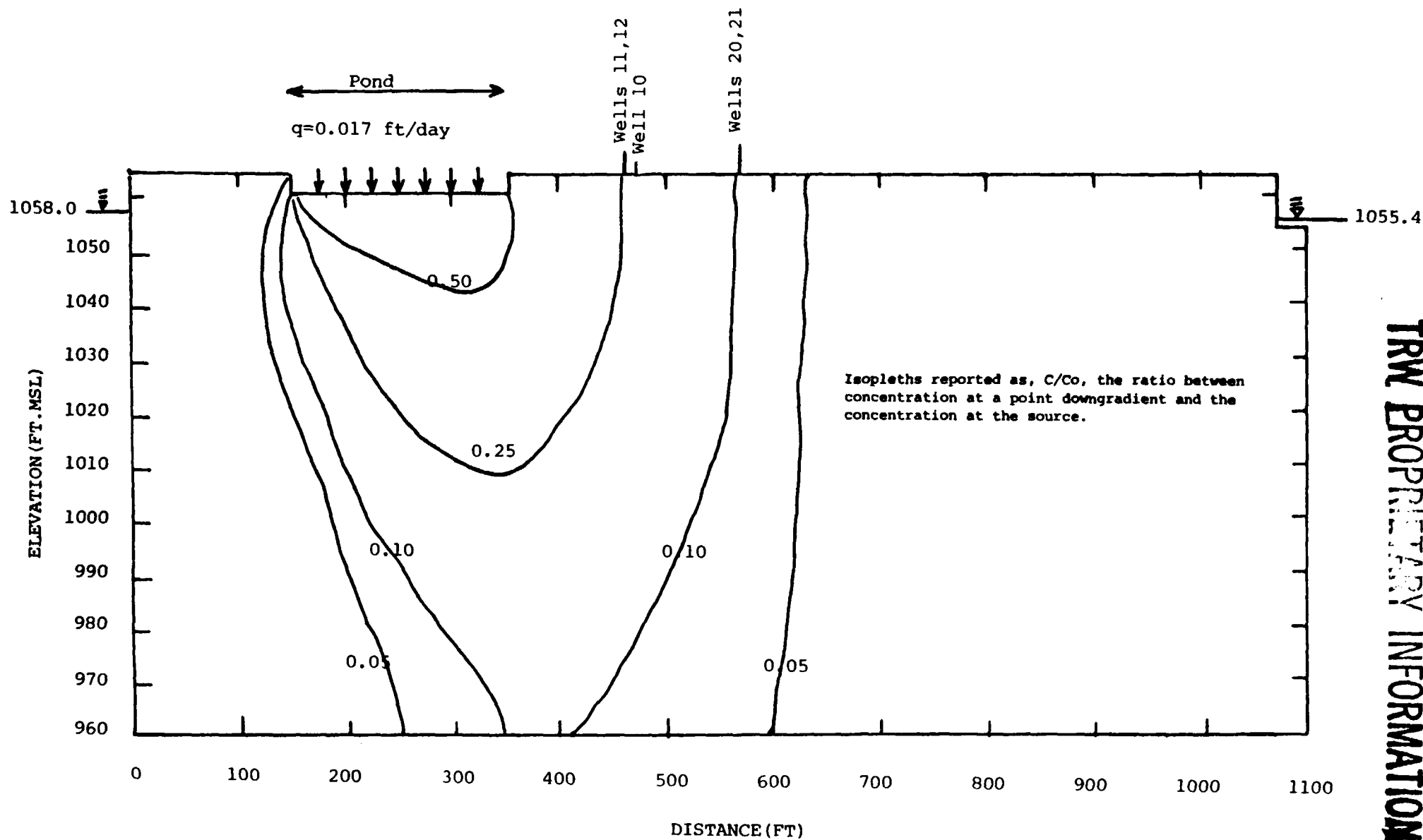
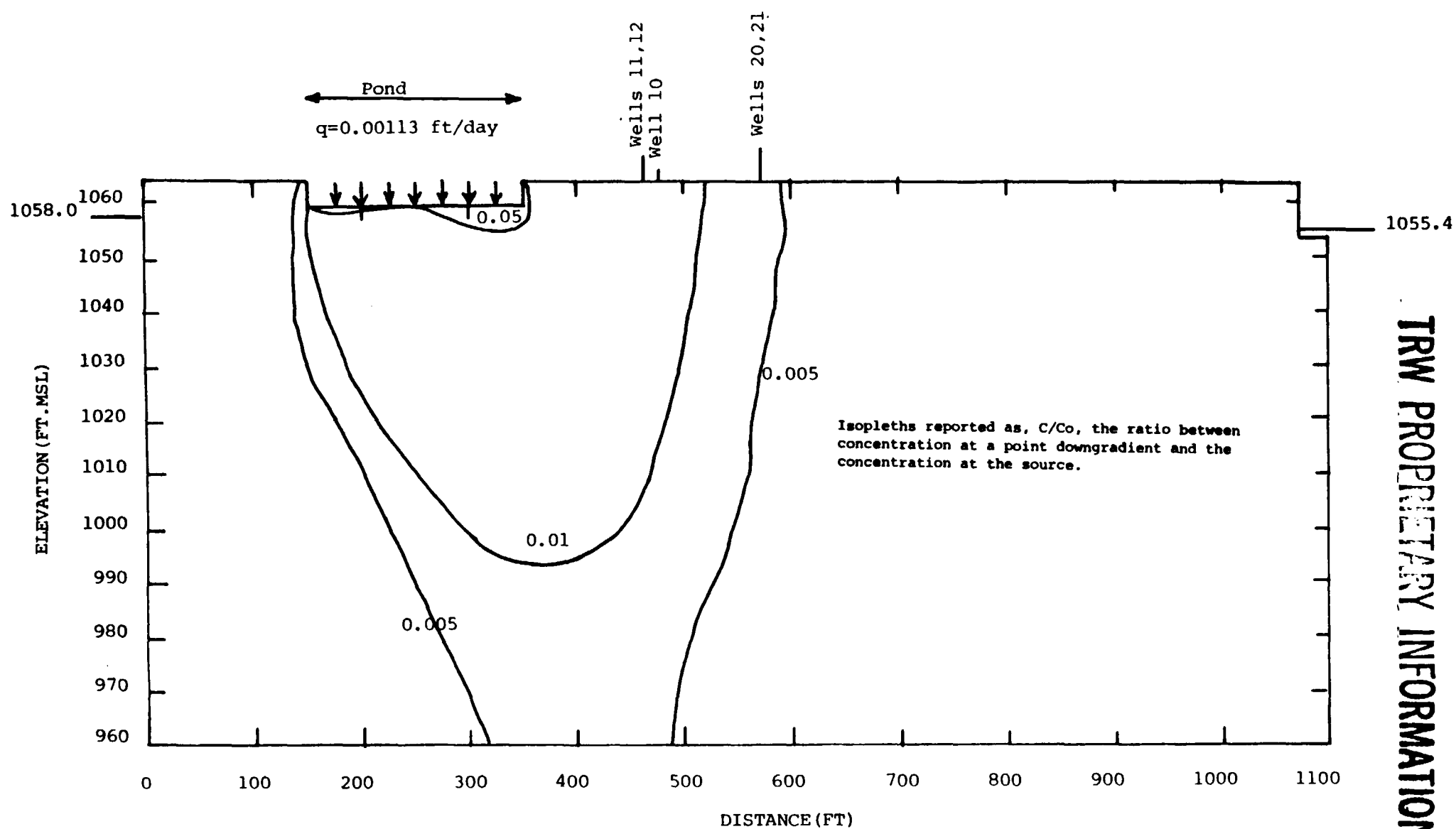


FIGURE B.6

VERTICAL MODEL: LOW FLOW

CONTAMINANT DISTRIBUTION AT 25 YEARS



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## REFERENCES

- OGATA, H., and BANKS, R.B. 1961. A Solution of the Differential Equation of Longitudinal Dispersion in the Porous Media. U.S. Geological Survey. Professional Paper 411-A
- U.S. DEPARTMENT OF AGRICULTURE (USDA). 1971. Soil Survey: Stark County, Ohio. USDA, Soil Conservation Service, in cooperation with Ohio Department of Natural Resources, Division of Lands and Soil, and Ohio Agricultural Research and Development Center. U.S. Government Printing Office
- YEH, G.T., and WARD, D.S. 1980. FEMWATER: A Finite Element Model of Water Flow Through Saturated-Unsaturated Porous Media. Oak Ridge National Laboratory. Report No. ORNL-5567. October 1980
- YEH, G.T., and WARD, D.S. 1981. FEMWASTE: A Finite Element Model of Waste Transport Through Saturated-Unsaturated Porous Media. Oak Ridge National Laboratory. Report No. ORNL-5601. April 1981

# TRW PROPRIETARY INFORMATION

## APPENDIX C

### SCIENTIFIC REVIEWS OF LITERATURE ON PCBs AND RELATED COMPOUNDS

- DRILL, FRIESS, HAYS, LOOMIS, and SCHAFFER, INC. 1982. Potential Health Effects in the Human from Exposure to Polychlorinated Biphenyls and Related Impurities. Arlington, Virginia
- ECOLOGY AND ENVIRONMENT, INC. 1981. Summary of the Health Effects of PCBs. Prepared for Chemical Manufacturers Association. Buffalo, New York
- HAMMOND, P.B., NISBET, I.C.T., and SAROFIM, A.F. 1972. Polychlorinated biphenyls: Environmental Impact. Env. Res. 5:249-362
- HIGUCHI, K. 1976. PCB Poisoning and Pollution. Academic Press, New York
- HUFF, J.E., MOORE, J.A., SARACCI, R., and TOMATIS, L. 1980. Long-term hazards of polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Environ. Health Perspec. 36:221-238
- HUTZINGER, O., SAFE, S., and ZITKO, V. 1979. The Chemistry of PCBs. 2nd Ed. CRC Press, Boca Raton, Florida
- HUTZINGER, O., et al. 1982. Chlorinated dioxins and related compounds: Impact on the environment. Pergamon Press, Oxford and New York
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER. 1978. Long-term hazards of polychlorinated dibenzodioxins and polychlorinated dibenzofurans. IARC Inten. Tech. Rep. No. 78/001
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER. 1978. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 18: Polychlorinated Biphenyls and Polybrominated Biphenyls. World Health Organization, Lyon, France
- JAMES, R.C., CRANMER, M.F., and HARBISON, R.D. 1981. Technical Review of the Health Effects of PCBs. Ecology and Environment, Inc. Buffalo, New York
- KAMRIN, M., and D'ITRI, F. 1983. PCBs: Human and Environmental Hazards. Ann Arbor Science Publications
- KHAN, M.A.Q., and STANTON, R.H. 1981. Toxicology of Halogenated Hydrocarbons: Health and Ecological Effects. Pergamon Press, New York



# TRW PROPRIETARY INFORMATION

- KIMBROUGH, R.D. 1974. The toxicity of polychlorinated polycyclic compounds and related chemicals. CRC Crit. Rev. Toxicol. 2:445-498
- KIMBROUGH, R.D., ed. 1980. Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products. Amsterdam, Elsevier/N. Holland Biomedical Press
- KIMBROUGH, R.D., BUCKLEY, J., FISHBEIN, L., FLAMM, G., KASZA, L., MARCUS, W., SHIBKO, S., and TESKE, R. 1978. Animal toxicology. Environ. Health Perspect. 24:173-184
- NATIONAL ACADEMY OF SCIENCES. 1979. Polychlorinated Biphenyls. Washington, D.C.
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH. 1977. Criteria for a Recommended Standard--Occupational Exposure to Polychlorinated Biphenyls. Washington, D.C. DHEW Publication No. (NIOSH) 77-225
- NATIONAL RESEARCH COUNCIL OF CANADA. 1978. Polychlorinated Biphenyls: Biological Criteria for an Assessment of Their Effects on Environmental Quality. NRC No. 11077
- NEW YORK ACADEMY OF SCIENCES. 1979. Health effects of halogenated aromatic hydrocarbons. Nicholson, W.J., and Moore, J.A., eds. Ann. N.Y. Acad. Sci. 320
- PARKINSON, A., and SAFE, S. 1981. Aryl hydrocarbon hydroxylase induction and its relationship to the toxicity of halogenated aryl hydrocarbons. Toxicol. Environ. Chem. Reviews 4:1-46
- PEAKALL, D.B. 1975. PCBs and their environmental effects. CRC Crit. Rev. Environ. Contam. 5:469-532
- U.S. DEPARTMENT OF ENERGY. 1978. Toxicology of Energy-Related Pollutants. Dept. of Energy Symp. Series 47
- U.S. ENVIRONMENTAL PROTECTION AGENCY. 1976. Proceedings of the National Conference on Polychlorinated Biphenyls. Chicago, Illinois, EPA 500/6-75-004
- U.S. ENVIRONMENTAL PROTECTION AGENCY. 1980. Ambient Water Quality Criteria for Polychlorinated Biphenyls. EPA 440/5-80-068
- WORLD HEALTH ORGANIZATION. 1979. Environmental Health Criteria for Polychlorinated Biphenyls and Polychlorinated Terphenyls. Geneva, Switzerland

# TRW PROPRIETARY INFORMATION

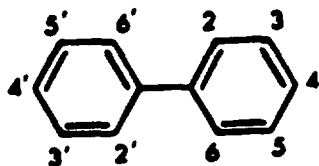
## APPENDIX D

### SUMMARY OF THE TOXICITY OF POLYCHLORINATED BIPHENYLS

This Appendix summarizes the scientific information that is most relevant to the assessment of the possible risks posed by exposure to low levels of PCBs in the environment. It is divided into three parts. The first summarizes the chemistry of PCBs and described the nature of the commercial mixtures, including the presence of highly toxic trace contaminants. The second summarizes the most relevant information on the toxicity of PCBs, using information both from epidemiological studies and from experimental studies in laboratory animals. The third summarizes the available information on the toxicity of the toxic contaminants. Together these sections summarize the most important information on the known effects of PCBs, illustrate the complexity of the problem of risk assessment, and document the statements made in the text and in Appendix E.

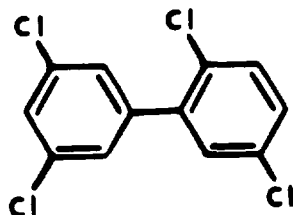
#### I. Nature and Composition of PCB Mixtures

Commercial polychlorinated biphenyls (PCBs) are complex mixtures of chlorobiphenyls with varying degrees of chlorination. As indicated below, the parent biphenyl molecule has ten positions at which chlorine substitution can occur.



# TRW PROPRIETARY INFORMATION

When the number but not the location of chlorine substitutions in a particular chlorobiphenyl is known, the chlorobiphenyl is referred to as a "congener" or "homolog" and is designated by a numerical prefix, e.g., tetrachlorobiphenyl. When the exact location of the chlorine substituents is known, the substance is referred to as an "isomer" and is identified by means of the numbering system indicated in the figure above. For example



is 2,3',5,5'-tetrachlorobiphenyl, one of 42 possible specific isomers of tetrachlorobiphenyl. Altogether there are 209 possible chlorobiphenyl isomers, at least 70 of which have been identified in commercial PCB mixtures.

Commercial production of PCBs involved chlorination of biphenyl with anhydrous chlorine in the presence of a catalyst which may be iron filings or ferric chloride, followed by treatment with alkali and distillation to purify the product. Depending on the process conditions, the product was a more or less complex mixture of chlorobiphenyls which was generally identified by the approximate percentage of chlorine it contained. In the case of the Aroclors formerly produced by Monsanto, the last two digits of the numerical designation (except for Aroclor 1016) indicated the approximate chlorine content; for example

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Aroclor 1254 contained about 54% chlorine and Aroclor 1242 contained about 42% chlorine. Other PCB manufacturers used different designations; for example the French Phenoclor DP6, and the German Clophen A60, like Aroclor 1260, contained about 60% chlorine. Other commercial PCB mixtures included the Kanechlors formerly produced in Japan and the Fenclores formerly produced in Italy.

The molecular composition of several different PCB mixtures has been investigated in several studies (see IARC 1978). In one such study by Webb and McCall (1973), the approximate composition of several Aroclors was determined as indicated in Table D.1. There is evidently considerable overlap in composition between different Aroclor mixtures.

In addition to chlorobiphenyls, commercial PCBs may contain small quantities of impurities. Of particular importance are the highly toxic chlorinated dibenzofurans (CDFs) first identified as impurities in PCBs by Vos et al. (1970). CDFs have been detected at levels of 1-2 ppm in several Aroclors (Bowes et al. 1975a), at up to 13.6 ppm in Phenoclor DP-6 (Bowes et al. 1975a), and at up to 18 ppm in Kanechlor 400 (Nagayama et al. 1976). In a more recent study by Morita et al. (1977), 16 different Japanese and European PCB mixtures were analyzed and found to contain 1-16 ppm of CDFs (Table D.2). Of particular interest was the finding that a sample of a Japanese PCB mixture (Aroclor T1248) contained 2.8 ppm CDFs while Aroclor T1248 which had been used for 2 years in a heat exchange unit had

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TABLE D.1

PERCENTAGE OF EACH CONGENER IN SEVERAL AROCLORS

Number of Chlorines	Aroclor					
	1221	1232	1242	1248	1254	1260
0	7	6				
1	51	26	1	.		
2	38	29	17	1		
3	3	24	40	23		
4		15	32	50	16	
5		0.5	10	20	60	12
6			0.5	1	23	46
7					1	35
8						6
9						
10						

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TABLE D.2

CONCENTRATIONS (ppm) OF CDFs  
OF DIFFERENT DEGREES OF CHLORINATION  
IN PCB SAMPLES AND "YUSHO OIL"

PCB	3-Cl	4-Cl	5-Cl	6-Cl	Total	Reference
Aroclor 1248	--	0.5	1.2	0.3	2.0	1
Aroclor 1254 <sup>a</sup>	--	0.1	0.2	1.4	1.7	1
Aroclor 1254 <sup>b</sup>	--	0.2	0.4	0.9	1.5	1
Aroclor 1260 <sup>a</sup>	--	0.1	0.4	0.5	1.0	1
Aroclor 1260 <sup>b</sup>	--	0.2	0.3	0.3	0.8	1
Aroclor 1016	--	<0.001	<0.001	<0.001	--	1
Aroclor T64	--	4.8	9.4	2.0	16.2	2
Aroclor T241	--	2.4	2.7	0.8	5.9	2
Aroclor T1242	--	2.3	2.2	--	4.5	2
Aroclor T1248	--	0.5	2.3	--	2.8	2
Aroclor T1248 <sup>c</sup>	0.3	5.8	5.6	0.7	12.4	2
Aroclor T1254	--	0.1	3.6	1.9	5.6	2
Aroclor T1260	--	0.8	0.9	0.5	2.2	2
Kanechlor KC300	--	6.7	1.6	--	8.3	2
Kanechlor KC400	0.3	12.2	10.4	0.9	23.8	2
Kanechlor KC500	0.2	1.7	1.1	3.1	6.1	2
Kanechlor KC600	--	0.2	0.5	0.4	1.1	2

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TABLE D.2, (continued)

PCB	3-C1	4-C1	5-C1	6-C1	Total	Reference
Clophen A-30	1.6	2.3	1.0	--	4.9	2
Clophen A-40	1.5	5.4	6.9	--	13.8	2
Clophen A-50	0.7	8.3	4.1	1.8	14.9	2
Phenoclor DP-4	--	1.7	1.6	0.5	3.8	2
Phenoclor DP-5	--	4.6	2.7	2.6	9.9	2
Phenoclor DP-6 <sup>a</sup>	0.2	2.1	2.6	5.6	10.5	2
Phenoclor DP-6 <sup>b</sup>	--	0.7	10.0	2.9	13.6	1
Yusho oil	0.02	0.5	1.3	0.8	2.7	2

<sup>a,b</sup> Different samples

<sup>c</sup> Used PCB (2 years in heat exchanger)

SOURCE: Bowes et al. 1975a, Morita et al. 1977

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12.4 ppm CDFs. Buser et al. (1978a) analyzed the same used PCB mixture and found slightly higher levels of CDFs (15.6 ppm).

High levels of CDFs were also found in the contaminated rice oil responsible for the disease known as Yusho. This disease occurred in Western Japan in 1968 among individuals who had consumed rice oil which had been contaminated by PCBs from a leaking heat exchanger. Estimates of the amount of CDFs in the "Yusho oil" have ranged from 2.68 ppm (Morita et al. 1977) to 5 ppm (Nagayama et al. 1976) and 5.6 ppm (Buser et al. 1978a). This corresponds to about 0.5% of the amount of PCBs in the Yusho oil. The high levels of CDFs in used PCBs may be the result of thermal decomposition of the PCB since CDFs are formed if PCBs are heated in air at temperatures above 270°C (Buser et al. 1978b, Morita et al. 1978). In addition to the PCBs and CDFs, the rice oil responsible for the Yusho incident also contained over 800 ppm of polychlorinated quaterphenyls (PCQs) (Miyata et al. 1978); polychlorinated quaterphenyl ethers (PCQEs) have also been identified in the rice oil (Miyata et al. 1979). Like CDFs, PCQs are formed when PCBs are heated, and up to 31,000 ppm of PCQs has been found in used PCB mixtures (Miyata et al. 1978). Very little is known about the toxicity of PCQs.

The presence of CDFs in PCB mixtures complicates interpretation of toxicology studies since CDFs are generally more toxic than PCBs (see Section III). However, the presence of CDFs



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cannot be ignored, particularly if the finding of higher levels of CDFs in used than in unused PCBs is of general applicability.

## II. Toxicity of PCBs

The toxicology of PCBs has been extensively reviewed in the past. Appendix C lists 25 reviews published since 1972. The following description of major toxicological findings is based upon these reviews, but emphasizes recent important studies.

### A. Chloracne and Yusho

Current knowledge of the effects of PCB exposure in humans comes largely from cases of accidental contamination of food by PCBs and from cases of occupational exposure to PCBs. The effects of occupational exposure to PCBs have been reviewed in detail by NIOSH (1977). The classic symptom of occupational PCB exposure is chloracne, a disfiguring skin disease characterized by small dermal cysts, pustules and comedones, most commonly on the face, ears, and neck, but also on areas in contact with contaminated clothing. However, chloracne is a symptom of systemic poisoning and not just the result of skin contact since symptoms of chloracne occurred in cases of eating contaminated food in the Yusho incident in Japan (Kuratsune et al. 1972) and in a similar case in Taiwan (Chang et al. 1980a, 1980b, 1981). NIOSH (1977) concluded that chloracne may occur in individuals exposed to PCB vapor concentrations as low as  $0.1 \text{ mg/m}^3$  for several months, or with blood PCB levels of about 200 ppb or more. Other effects that have been attributed to

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occupational exposure to PCBs include digestive disturbances, eye irritation, liver injury, and impotence (NIOSH 1977).

More recently, Warshaw et al. (1979) reported an unusual incidence of abnormalities in pulmonary function in a group of capacitor-manufacturing workers exposed to PCBs. Among 243 workers with no history of exposure to asbestos, talc, or textile fibers which might affect pulmonary function, 34 (14%) showed impaired forced vital capacity (FVC). Impairment was defined as an age- and sex-specific FVC less than 79.5% of that predicted by standard values for healthy nonsmoking adults.

The incidence of impaired FVC among the capacitor workers (34/243, 14%) was similar to that seen by the authors among asbestos insulation workers (22/182, 12.1%) and greater than among nonsmoking adults (54/957, 5.5%). Among the 34 workers with impaired FVC, 27 had restrictive impairment but only one of these had radiographic abnormalities on a chest X-ray. The authors noted that restrictive impairment without radiographic changes is unusual in occupational exposures and that a reduction in FVC is not a characteristic change in cigarette smokers except when accompanied by generalized airway obstruction, rather than restriction. This finding of impaired pulmonary function among workers exposed to PCBs deserves further study.

Alvares et al. (1977) found that the plasma half-life of aminopyrine in five workers exposed to Aroclor 1016 for 2 years and other Aroclors prior to that was shorter than the

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plasma half-life in a control group. Since aminopyrine is metabolized by the hepatic mixed function oxidase (MFO) system, the shorter half-life indicates these hepatic enzymes were induced in the exposed workers.

PCBs present in human mothers are transferred to babies via the milk (Kuwabara et al. 1978). Mothers (N=20) occupationally exposed to Kanechlor 300 and/or Kanechlor 500 had PCB blood levels of 8.3 to 84.5 ppb. while their children (N=39) had blood levels of 0.8 to 93.2 ppb. The PCB levels in the children aged 0-13 years were directly related to the length of time each was nursed. Infants not breast-fed by exposed mothers had PCB blood levels lower than their mothers'. Gas chromatographic analysis of mother and child's blood showed the same general peak pattern, but the peaks were smaller in the children. The maternal exposure or absorption of PCB was not quantified.

Two major incidents of dietary exposure to PCBs have been reported. The first occurred in 1968 in western Japan. The initial investigations of this disease have been reviewed by Kuratsune et al. (1972). The symptoms first reported were a chloracne-like skin disease which was given the name Yusho (oil disease) when the cause was identified as a particular brand of rice oil that had been contaminated during manufacture by a leaking heat exchanger. Recent estimates indicate that the most contaminated batch of rice oil contained about 1000 ppm PCBs, 5 ppm CDFs, and 866 ppm PCQs (Hayabuchi et al. 1979);

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the presence of PCQEs has also been reported (Miyata et al. 1979). More than 1,000 individuals were affected by the disease. Common symptoms are indicated in Table D.3 which is taken from Kuratsune et al. (1972).

A dose-response relationship was noted between the amount of contaminated oil used and the severity of the disease as indicated in Table D.4 (Kuratsune 1972). More recently Hayabuchi et al. (1979) estimated the oil consumption of 72 Yusho victims and determined that the severity of the symptoms correlated well with the total oil consumption but not with the daily consumption on a  $\mu\text{l/kg/day}$  basis. These authors estimated that the average total amounts of PCBs, CDFs, and PCQs consumed by Yusho victims were 466, 2.5, and 439 mg, respectively, while the smallest total amounts consumed by symptomatic individuals were 111, 0.6, and 105 mg respectively, corresponding to daily doses of 29, 0.16, and 27  $\mu\text{g/kg/day}$ , respectively.

Symptoms of the disease were seen in babies born to women exposed to PCBs either during pregnancy or during breast feeding. The babies exposed in utero had grayish, dark-brown stained skin at birth, most had increased eye discharge, and several were small-for-date. One baby born before its mother was exposed apparently contracted the disease subsequently through breast feeding (Kuratsune et al. 1976). One stillborn fetus showed marked hyperkeratosis and atrophy of the epidermis, and systic dilation of hair follicles, especially on the head (Kuratsune et al. 1972).

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TABLE D.3

DISTRIBUTION OF SYMPTOMS REPORTED BY 189 YUSHO VICTIMS  
EXAMINED BEFORE OCTOBER 31, 1968

Symptoms	Females (N=89) (%)	Males (N=100) (%)
Dark brown pigmentation of nails	83.1	75.0
Distinctive hair follicles	64.0	56.0
Increased sweating at palms	50.6	55.0
Acnelike skin eruptions	87.6	82.0
Red plaques on limbs	20.2	16.0
Itching	42.7	52.0
Pigmentation of skin	75.3	72.0
Swelling of limbs	20.2	41.0
Stiffened soles in feet and palms of hands	24.7	29.0
Pigmented mucous membrane	56.2	47.0
Increased eye discharge	88.8	83.0
Hyperemia of conjunctiva	70.8	71.0
Transient visual disturbance	56.2	55.0
Jaundice	11.2	11.0
Swelling of upper eyelids	71.9	74.0
Feeling of weakness	58.4	52.0
Numbness in limbs	32.6	39.0
Fever	16.9	19.0
Hearing difficulties	18.0	19.0
Spasm of limbs	7.9	8.0
Headache	30.3	39.0
Vomiting	23.6	28.0
Diarrhea	19.1	17.0

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TABLE D.4

RELATION BETWEEN AMOUNT OF RICE OIL USED  
AND CLINICAL SEVERITY OF YUSHO

Amount of oil used	Unaffected		Light cases		Severe cases		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Less than 720 ml	10	(12)	39	(49)	31	(39)	80	(100)
720-1440 ml	0	(0)	14	(31)	31	(69)	45	(100)
More than 1440 ml	0	(0)	3	(14)	18	(86)	21	(100)

These authors also reported that after exposure had ceased, during the period between summer 1969 and 1970, of 159 patients examined 81 had improved, 20 had worsened, and 58 had remained unchanged, and that even among those who had improved many still had serious complaints including persistent headache, general fatigue, weakness, numbness of the limbs, and weight loss (Kuratsune et al. 1972). Some patients displayed a peripheral neuropathy that was selective for sensory nerves (Murai and Kuroiwa 1971). By 1974, the skin symptoms had generally improved but symptoms such as general fatigue, poor appetite, headache, numbness and pain in the legs, cough and expectoration of sputum continued to be prominent for at least 10 years after exposure (Kuratsune et al. 1976, Urabe et al. 1979). Kuratsune et al. (1976) also reported a high incidence of menstrual disorders among Yusho

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victims in 1974. The respiratory symptoms (cough and expectoration) were frequently associated with shadows on chest X-rays, and among 12 patients with respiratory distress who were studied in detail, 5 had chronic viral or bacterial airway infection suggesting reduced resistance to infection, possibly associated with the known effects of PCBs on the immune system (Shigematsu et al. 1978). The affected babies born to mothers exposed during pregnancy improved as they grew older (Kuratsune et al. 1972, 1976) and have not displayed any neurobehavioral effects as have been seen in monkeys and mice exposed during gestation and via breast milk.

The second major incident of contamination of food with PCBs also affected more than 1,000 persons and occurred under similar circumstances in central western Taiwan in 1979 (Chang et al. 1980 a,b, 1981). Once again contamination of rice bran oil by PCBs was responsible and a similar spectrum of symptoms was noted; acne-like skin eruptions, pigmentation of the nails, swelling of the eyelids and increased eye discharge (hypersecretion of Meibomian gland), headache, nausea, and numbness of the limbs. In addition to the effects seen in the Yusho incident, liver damage, immunologic impairment, and increased urinary excretion of heme precursors were noted. These effects had previously been observed in animals (see below). Liver damage was indicated by increased activities of the enzymes SGOT, SGPT, and alkaline phosphatase in blood serum (Chang et al. 1980a). The individuals exposed to PCBs had lower serum

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$\gamma$ -globulin levels and displayed decreased delayed-type-hypersensitivity responses to streptokinase and streptodornase (Chang et al. 1980a). These individuals also had decreased levels of immunoglobulins IgA and IgM in their serum, and lower percentages of T cells, especially helper T cells, among their lymphocytes (Chang et al. 1981). Chang et al. (1980b) examined the urinary excretion of heme precursors in 69 patients exposed to PCBs and 20 normal individuals. Significantly increased levels of  $\delta$ -aminolevulinic acid and uroporphyrin were noted in the 24-hour urine of the PCB-exposed individuals compared to the normal subjects. No effect on urinary excretion of porphobilinogen or coproporphyrin was noted. Reports of the Taiwan incident have not mentioned CDFs, PCQs, or other contaminants.

In a recent epidemiological study of 458 members of a community in Alabama with no unusual exposure to PCBs, except that from a high intake of fish caught locally, a significant ( $p < 0.05$ ) positive correlation between serum PCB level (range 3.2-157.4  $\mu\text{g/liter}$ , geometric mean 17.2  $\mu\text{g/liter}$ ) and diastolic blood pressure in adults was noted (Kreiss et al. 1981). Positive correlations were also observed between serum PCB concentration and gamma-glutamyl transpeptidase (GGTP) serum level, an indicator of liver disfunction, and between PCB level and serum cholesterol level. This study is of particular importance since it suggests the occurrence of subtle adverse effects resulting from exposure to relatively low ambient levels of PCBs. In a study of a smaller number of individuals (148)



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with varying degrees of exposure to PCBs, Baker et al. (1980) also observed a correlation between serum PCB and GGTP levels; however, this correlation was eliminated when alcohol drinkers were excluded from the analysis. Baker et al. also noted a positive correlation between PCB level and serum triglyceride level as had been seen in Yusho victims in Japan (Kuratsune 1972); this correlation remained significant when analysis was limited to nondrinkers. However, it is not clear whether these associations between PCB levels in the blood and indicators of cardiovascular risks would indicate a cause-and-effect relationship. Since PCBs are associated with lipids in the blood, it is possible that individuals with elevated serum triglycerides would have elevated PCB levels for that reason.

## B. Evidence of Carcinogenicity in Humans

There is no direct evidence that PCBs are carcinogenic in humans. However, Unger and Olsen (1980) have noted a statistically significantly ( $p < 0.01$ ) greater concentration of PCBs and DDE (a metabolite of the insecticide DDT) in adipose tissue of terminal cancer patients with a wide variety of types of cancer, compared to that of patients who died of other diseases, when the statistics were adjusted for age and sex. Also, Wassermann et al. (1978) observed higher levels of PCBs in the tissues of patients with gastric carcinoma than in the controls ( $p < 0.05$ ); and within the tissues of the cancer patients, the level of PCBs was higher in the tumor tissue than in adjacent normal

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gastric mucosa ( $p < 0.01$ ). However, similar associations were observed with DDE and other organochlorines, and these findings are therefore ambiguous.

In a retrospective cohort mortality study of PCB-exposed workers making electrical capacitors (Brown and Jones 1981), mortality for all causes was lower than expected on the basis of U.S. age- and sex-adjusted, cause-specific mortality rates (163 observed, 182.4 expected). Mortality from all types of cancer combined was also lower than expected (39 observed, 43.8 expected). There was, however, a slight but nonsignificant excess of rectal cancer (4 observed, 1.19 expected) and liver cancer (3 observed, 1.07 expected), and in one plant there was a slight excess of deaths due to cirrhosis of the liver (5 observed, 3.2 expected). The latter two effects may be consistent with the known hepatotoxic effects of PCB, but the numbers involved are too small for any reliable conclusions to be drawn.

Bahn et al. (1976) reported an excess of melanomas (2 observed, 0.04 expected) among 31 men previously exposed to PCB in the research laboratory of a petrochemical plant. However, the extent of exposure to other potential carcinogens was not reported. Hence the cause of the melanomas is not clear.

A somewhat elevated incidence of death due to cancer has also been noted among Yusho victims. Among 31 deaths whose causes were confirmed up to 1977, 11 were from neoplasms (35.4% of the total). This fraction is higher than that observed

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(21.1%) for deaths due to neoplasms among inhabitants of the same area of Japan in 1977 (Urabe et al. 1979). However, this comparison was not standardized for age or other differences between the two populations. Among the cancer deaths were 2 with stomach cancer, 1 with both stomach and liver cancer, 2 with liver cancer and liver cirrhosis, and 3 with tumors of the lung.

## C. Effects Observed in Experimental Animals

Many of the effects of PCB exposure seen in humans are also observed in animals, and in many cases these effects can be studied more easily in animals than in humans. In addition, there are effects seen in animals that may potentially occur in humans; but evidence for their occurrence in humans is lacking, especially carcinogenicity and mutagenicity. The major effects of repeated exposure of animals to PCBs are briefly summarized below.

### 1. Carcinogenicity

There is evidence from several studies to implicate PCBs in the production of liver tumors in rats and mice, and there is some evidence to suggest that PCBs may also induce tumors of the stomach and lower gastrointestinal tract in rats.

Kimbrough et al. (1975) fed Aroclor 1260 at 100 ppm in the diet of female Sherman rats for 21 months. Among the 184 surviving treated rats, 26 had hepatocellular carcinoma and an additional 144 had neoplastic nodules in the liver. Among

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173 surviving control animals, only 1 had liver carcinoma and none had neoplastic nodules.

The National Cancer Institute (1978) performed a carcinogenesis bioassay on Aroclor 1254. Groups of 24 male and 24 female Fisher 344 rats were fed diets containing 0, 25, 50, and 100 ppm Aroclor 1254 for 104-105 weeks. There was a small, dose-related increase in the incidence of liver hepatocellular adenoma and carcinoma and a larger increase in the incidence of nonneoplastic liver nodular hyperplasia as shown in Table D.5 below.

TABLE D.5  
INCIDENCE OF LIVER LESIONS IN RATS IN NCI BIOASSAY

	Control	25 ppm	50 ppm	100 ppm
Hepatocellular adenoma and carcinoma (males)	0/24	0/24	1/24	3/24
Hepatocellular adenoma and carcinoma (females)	0/23	0/24	1/22	2/24
Nodular hyperplasia (males)	0/24	5/24	8/24	12/24
Nodular hyperplasia (females)	0/23	6/24	9/22	17/24

If males and females are combined, the incidence of hepatocellular adenoma and carcinoma is significantly greater in the 100 ppm group than in the control group ( $p=0.030$ , Fisher's exact test). In addition to these liver lesions, there was

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a total of four adenocarcinomas and one carcinoma in the gastrointestinal tract of treated rats but none in the controls. These are uncommon tumors in this strain of rats. The historical incidence of such tumors is 6/600 in males and 2/600 in females. NCI concluded that these tumors may have been related to the Aroclor 1254 treatment. The incidence of hepatocellular adenoma and carcinoma seen at 100 ppm in this study (10.4%) is consistent with the incidence (14.1%) observed in the study by Kimbrough et al. (1975), but the high incidence of neoplastic nodules seen in the Kimbrough et al. (1975) study was not seen in the NCI bioassay. A high incidence of nodular hyperplasia was seen, however. Nodular hyperplasia is considered an early, preneoplastic lesion which may progress to neoplasia under suitable circumstances (Pitot and Sirica 1980). Thus the main difference between the results of the two experiments is that the tumors developed more slowly in the NCI study.

Kimbrough and Linder (1974) also reported that Aroclor 1254 fed to BALB/cJ male mice at 300 ppm in the diet for 11 months induced hepatomas in 9/22 surviving animals. No hepatomas were seen in the livers of 58 mice fed a normal diet for 11 months. The PCB-fed mice also had greatly enlarged livers and adenofibrosis of the liver.

## 2. Interactions with Other Carcinogens

Early studies of interaction indicated that treatment of animals with certain carcinogens and with PCBs at the same time delayed or inhibited the action of the carcinogen. For

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example, Makiura et al. (1974) observed a greatly reduced incidence of liver tumors in rats fed PCB at the same time as 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB), N-2-fluorenylaceta-mide (2-FAA) or diethylnitrosamine (DENA). The authors speculated that the inhibitory effects may be attributed to stimulation of microsomal enzymes by PCB, enhancing metabolism of the carcinogens.

More recently, several studies have indicated that when PCBs are administered to rats following a low dose of a liver carcinogen, the incidence of liver tumors is greatly enhanced by the PCB treatment. This promoting effect has been observed with DENA (Nishizumi 1976, 1979, Preston et al. 1981), 2-FAA (Ito et al. 1978, Tatematsu et al. 1979), and 3'-Me-DAB (Kimura et al. 1976). In the study by Preston et al. (1981), for example, groups of 40 male Sprague-Dawley rats were given drinking water containing DENA at 66 µg/ml for 5 weeks, and then Aroclor 1254 was added to the diet of one group for a further 18 weeks. An additional group of 40 did not receive DENA but were given Aroclor 1254 in weeks 6-23. An untreated group was also included. In rats surviving to 23 weeks, none of the control rats or rats receiving only Aroclor had liver tumors. Of 32 rats receiving only DENA, 5 (16%) had hepatocellular carcinomas, while 21/33 (64%) of the rats given DENA followed by Aroclor had hepatocellular carcinomas. In the study by Kimura et al. (1976), Kanechlor 400 at 400 ppm in the diet for 6 months enhanced the production of hepatocellular carcinoma in rats previously fed 3'-Me-DAB at 600 ppm in the diet for 2 months. However,

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the incidence of carcinoma was reduced if Kanechlor was given before or concurrently with 3'-Me-DAB. Also, when rats were exposed in utero and via their mothers' milk to PCBs (Kanechlor 500), they were less sensitive than unexposed rats to subsequent induction of liver tumors by DENA (50 ppm for 5 weeks after weaning), showing a significantly reduced ( $p < 0.05$ ) number of liver tumors per rat (1 tumor/rat in males, 0 in females) compared to rats not exposed to Kanechlor (3 tumors/rat in males, 1.1 in females) (Nishizumi 1980). The fact that increased tumor yield occurs only when PCBs are given after the carcinogen suggests that the effect is not simply related to the ability of PCBs to induce liver enzyme systems and increase metabolic activation of the carcinogen. Rather, PCBs appear to be acting as promoters. However, PCBs neither have promoting activity in the classical two-stage mouse skin tumorigenesis system using 7,12-dimethylbenz(a)anthracene as the initiator (Berry et al. 1978), nor in a system in which the cervical epithelium of mice was treated with 3-methylcholanthrene (MC) for 4 weeks and the mice given a diet containing 10 or 100 ppm Kanechlor 400 during and for 3 weeks after MC treatment (Uchiyama et al. 1974). However, these authors did not try administering PCBs only after treatment with the carcinogen, but used concurrent treatments.

### 3. Mutagenicity

Most studies of the mutagenicity of PCBs have given negative results, including assays for reversion in Salmonella typhimurium

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(Hsia et al. 1978, Schoeny et al. 1979), chromosome aberrations in rat bone marrow (Green et al. 1975a), dominant lethal mutation in rats (Green et al. 1975b), and chromosome breakage in Drosophila (Nilsson and Ramel 1974). These studies used 2,2',5,5'-tetrachlorobiphenyl, Aroclors 1242 and 1254, and Clophens A30 and A50. However, another study has provided evidence that chlorinated biphenyls with a lower degree of chlorination may be mutagenic in Salmonella strain TA1538 in the presence of a rat liver metabolic activation system (Wyndham et al. 1976). Wyndham et al. (1976) also noted binding of  $^3\text{H}$ -4-chlorobiphenyl to protein and RNA when the chemical was added to a liver microsome preparation. In a later study, Wyndham and Safe (1978) presented evidence that 4-chlorobiphenyl was metabolized via an arene oxide intermediate. Arene oxide intermediates are the reactive derivatives believed to be involved in the binding of carcinogenic polynuclear aromatic hydrocarbons to DNA. It is likely that the arene oxide intermediate is responsible for the mutagenic activity of 4-chlorobiphenyl. Negative results were obtained with 4-chlorobiphenyl in a modified assay for mutagenicity in Salmonella (McMahon et al. 1979), but it is unclear what doses were tested or how the modifications used may have affected the sensitivity of the assay.

#### 4. Reproductive, Teratogenic, and Postnatal Effects

Exposure to PCBs at various times before, during and after pregnancy has resulted in various degrees and types of adverse effects on reproduction and on the offspring in many species.



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Among the species most sensitive to these effects of PCBs are the monkey and the mink, but all species that have been tested, including rats, mice, rabbits, and miniature swine, have shown effects. Studies with monkeys (Allen et al. 1979, 1980; Bowman et al. 1978, 1981) have examined the reproductive effects of PCBs and the redistribution of maternal PCBs to the fetus trans-placentally and to the nursing offspring via the milk.

Adult female rhesus monkeys received Aroclor 1248 at dietary levels of 0.5, 1.0, 2.5 or 5.0 ppm for 18 months. Prolonged menses and elevated serum progesterone levels were observed after 4 months of treatment. The females were mated with untreated males after 7 months of treatment and again 1 year after PCB treatment was stopped. The results of mating for the females in the 2.5 and 5.0 ppm group are shown in Table D.6 below.

TABLE D.6

REPRODUCTIVE PERFORMANCE OF MONKEYS EXPOSED  
TO POLYCHLORINATED BIPHENYLS IN THEIR DIETS FOR 18 MONTHS

	During PCB Exposure		1 Year After PCB Exposure	
	2.5 ppm	5.0 ppm	2.5 ppm	5.0 ppm
Total impregnated	8/8	6/8	8/8	7/7
Absorptions/resorptions	3/8	4/8	1/8	1/7
Stillborn	0/8	1/8	0/8	1/7
Normal births	5/8	1/8	7/8	5/7

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Infants in each treatment group in each mating had reduced body weights at birth. No gross external malformations were reported. After the first mating, the six infants were permitted to nurse for 4 months. Within 2 months focal areas of hyperpigmentation, swollen lips and eyelids, loss of eyelashes, and acneform lesions of the face developed in the infants. The skin of these infants showed a decided increase in the PCB level over this period. Within 4 months, three of the six infants died (one in the 5.0-ppm group and two in the 2.5-ppm group) due to PCB intoxication. After weaning, the remaining three showed improvement of the skin lesions during the subsequent 4-month period. At weaning and 2 years later, the surviving offspring of the first mating in the 0.5, 1.0, and 2.5-ppm groups were similarly hyperactive and had comparable deficits in learning ability. At 4 years of age these offspring were hypoactive (Bowman et al. 1981).

After the second mating, the breast milk of the mothers contained 0.02-0.19  $\mu\text{g}$  PCB/g whole milk, and the hairline of the infants showed hyperpigmentation. Two infants in each group died after weaning. These four infants showed hypocellularity of the thymus, grossly small spleens, and reduced number of lymph nodes. Histologically the lymph nodes were hypocellular and devoid of germinal centers, while the bone marrow was hypoactive as evidenced by the majority of cells being mature (Allen et al. 1980). These histologic findings are indicative of

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impairment of the immune system, an effect of PCBs which is discussed in more detail later.

Male monkeys appear to be less susceptible than females to reproductive dysfunction by PCBs (Allen and Norback 1976). Four adult male rhesus monkeys were given a diet containing 5.0 ppm Aroclor 1248 for 17 months (average total intake of PCBs 460 mg). They began to develop a slight periorbital edema after 6 months of exposure; however, it was much less severe than in the female monkeys receiving a similar level of PCB. The morphological features and viability of the spermatozoa, as well as the ability to fertilize control monkeys, was unaffected during the initial 12 months of PCB exposure. Subsequently, one of the four males lost weight and developed alopecia, acne, periorbital edema, and decreased libido. A testicular biopsy of this animal showed a decided hypoactivity of the seminiferous tubules. There was an absence of mature spermatozoa and a predominance of Sertoli cells of the tubules. The remaining three males remained healthy and sexually active (Allen and Norback 1976).

Reproduction in mink is also adversely affected by low levels of PCBs. The degree of reproductive impairment is different for different PCB mixtures. Aroclor 1254 at 2 ppm in the diet dramatically decreased the number of live kits born per female. Aroclor 1242 had a similar effect at 5 ppm in the diet. Aroclor 1016, however, had considerably less effect on reproduction. A dietary level of 2 ppm was without effect,

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while 10 ppm reduced the number of successful pregnancies but did not affect the number of kits per litter (Aulerich and Ringer 1977, 1980, Bleavins et al. 1980).

The teratogenic potential of PCBs, both individual isomers and commercial mixtures, has been studied in several species. In a study by Masuda et al. (1979), mice were fed diets containing known levels of each of seven chlorobiphenyl isomers (tri- to octachloro) for 18 days either before or during pregnancy.

Several isomers reduced the number offspring per female, but the only isomer that caused measurable defects in the offspring was 3,3',4,4'-tetrachlorobiphenyl. Offspring of mice treated with this chemical showed increased locomotor activity and general restlessness ("waltzing syndrome") within 2-3 weeks of birth. It could not be determined whether placental transfer or uptake of PCB from maternal milk was the critical route of exposure (Lucier et al. 1978).

Tilson et al. (1979) and Marks et al. (1981) also reported neurobehavioral defects in the offspring of female mice treated with 3,3',4,4'-tetrachlorobiphenyl and 3,3',4,4',5,5'-hexachlorobiphenyl during pregnancy. However, these isomers are only minor components in commercial PCB mixtures, so the relevance of these studies to risk assessment for environmental PCBs is limited.

Several studies of the effects of PCB mixtures have also been conducted. Villeneuve et al. (1971) reported that single oral doses of 12.5, 25, and 50 mg/kg Aroclor 1254 administered

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to pregnant rabbits were fetotoxic, but did not induce teratogenic effects. Linder et al. (1974) reported that Aroclors 1254 and 1260 had minor fetotoxic effects when administered at 100 mg/kg/day to female rats on days 7-15 of pregnancy; Aroclor 1254 also caused substantial mortality in pups prior to weaning.

Earl et al. (1974) reported that Aroclor 1254 caused reproductive impairment and skeletal abnormalities in the offspring of beagle dogs and miniature swine exposed during pregnancy to doses of 5 mg/kg/day and 10 mg/kg/day, respectively. However, this study was reported only in an abstract, and details are not available for review.

In summary, PCB mixtures have been reported to affect reproduction in a number of animal species, but rhesus monkeys and mink appear to be much more sensitive than other species. Teratogenic effects have not been convincingly reported, except for neurobehavioral effects in mice caused by isomers that are only very minor components in commercial mixtures.

## 5. Liver Toxicity

An increase in liver weight and/or in liver-to-body weight ratio in response to PCB treatment has been noted in many studies. Litterst et al. (1972) observed increased liver-to-body weight ratios in rats fed 50 and 500 ppm of Aroclors 1242, 1248, 1254, and 1260 for only 4 weeks. Similar increases in liver weight have been noted in long-term feeding studies with these mixtures in rats (Bitman et al. 1972; Linder et al. 1974). In addition,

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effects were noted at much lower dietary levels in offspring of treated rats (exposed during gestation and prior to weaning): significant increases in liver weight were noted in weanling rats after maternal exposure to only 5 ppm Aroclor 1260 and only 1 ppm Aroclor 1254 (Linder et al. 1974).

Increase in liver weight has been noted as an effect of PCBs in many other species (Vos 1972, Nishizumi 1970, Barsotti and Allen 1975, Hansen et al. 1975).

The initial increase in liver weight is due mainly to proliferation of smooth surfaced membranes of the endoplasmic reticulum, a network of interconnected channels present in the cytoplasm of most animal cells. Norback and Allen (1972) observed a proliferation of smooth endoplasmic reticulum (SER) in rats fed PCBs for one to five weeks. Similar proliferation of SER has been observed in mouse and monkey livers (McNulty 1976, Nishizumi 1970).

Changes in biochemical composition of the liver after exposure to PCBs include a significant increase in total lipid content (Grant et al. 1971). Triglyceride content of the liver increased with increased dosage of PCBs (from 5 to 500 ppm) in the diet (Litterst et al. 1972). This increase was greater with Aroclor 1248 than with 1242, 1254, or 1260. Norback and Allen (1972) found the ratio of phospholipids to protein increased and the ratio of cholesterol to protein increased. Hinton et al. (1978), on the other hand, found that although the total amount of liver phospholipid and cholesterol increased, the

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phospholipid/protein and cholesterol/protein ratios did not change when rats were treated with large doses of Aroclor 1254 (25 and 50 mg/kg). Both the total triglyceride and triglyceride/protein ratio increased markedly. They found the increase in phospholipids and triglycerides was caused by a decreased degradation or removal of the lipids from the liver and not an increase in synthesis. Marked increases in liver lipids have also been noted in treated rabbits (Ito et al. 1971).

## 6. Induction of Hepatic Mixed Function Oxidases

The hepatic mixed function oxidases (MFO) make up a membrane-bound enzyme system found mainly in the SER of the liver cells. MFO usually increases in activity with an increase in the SER. This enzyme system is able to metabolize and detoxify oxidatively a wide variety of exogenous and endogenous compounds. A major component of this system is a cytochrome enzyme, which because of its ability to absorb light, under specific conditions, with wavelengths around 450 nm, is called cytochrome P-450. Initially it was believed there was only one such enzyme, but it is now known that there is a family of these enzymes. Each cytochrome P-450 has its own enzymatic activities and spectral properties. In the normal animal not all the cytochrome P-450s are present. A variety of chemicals, such as PCBs, are, however, able to cause the synthesis of specific cytochromes and thus cause an increase in the total amount of enzyme and the enzymatic activity. This is considered induction of the MFO.

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Disruptions in normal hepatic MFO activity have been observed with PCBs even at dose levels which had no effect on liver weight or on liver-to-body weight ratios (Litterst et al. 1972). Several hepatic enzyme systems are affected in rats at dietary levels as low as 5 ppm (Chen and Dubois 1973) or even 0.5 ppm (Litterst et al. 1972), and "no-effect levels" have not yet been found. Application of very small amounts of PCBs to the skin of experimental animals can cause a marked increase in MFO activity (Kappas and Alvares 1975).

After termination of exposure, the enzyme-inducing effect of PCBs appears to be reversible. Litterst and Van Loon (1974) maintained rats on a high dietary concentration of 50 ppm PCB for seven days. Discontinuation of PCB treatment resulted in a slow decay of the induced enzyme activity to approximately control levels after 10 days. The various effects of PCBs on liver microsomal enzymes can be blocked by the prior administration of actinomycin D (Kimbrough 1974). Actinomycin D acts as an inhibitor of DNA-dependent RNA synthesis. Alvares et al. (1973) interpreted the inhibitory effect of actinomycin D on PCB-induced increases in enzyme activity as support for the view that PCBs enhance the synthesis of a distinct microsomal hemo-protein not present in liver of untreated rats.

There are two general classes of inducers of MFOs. These classes are characterized by the compounds phenobarbital (PB) and 3-methylcholanthrene (3-MC). The two compounds induce different species of cytochrome P-450. Ryan et al. (1979)



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isolated from rat liver the cytochrome P-450s induced by these compounds and by Aroclor 1254. Three cytochrome P-450s were isolated from treated rat liver and designated P-450a, P-450b, and P-450c. Phenobarbital induced P-450a and P-450c while 3-MC induced P-450a and P-450b. Molecular weight analysis, spectral properties, catalytic activity, reactivity with antibodies, and peptide mapping all showed that Aroclor 1254 induced three individual forms of P-450 and that they were identical to those induced by PB or 3-MC. Thus Aroclor 1254 has the characteristics of both PB and 3-MC for induction of MFOs.

A number of recent studies have started to elucidate structure-activity relationships in enzyme induction by chlorobiphenyls in rats (Ecobichon 1976, Poland and Glover 1977, Yoshimura et al. 1979, Goldstein 1979). The activity of PCB mixtures in inducing the specific enzymes characteristic of both PB and 3-MC type inducers has been shown to be caused by individual isomers that are either PB or 3-MC type inducers (Poland and Glover 1977, Goldstein et al. 1977, Goldstein 1979, Yoshimura et al. 1979). In the study by Yoshimura et al. (1979), the effect of individual isomers on the rat hepatic MFO activities of benzphetamine N-demethylase (BZ) and arylhydrocarbon hydroxylase (AHH) were measured as well as the concentration of cytochrome P-450. BZ activity, representative of PB type induction, was increased by specific PCB isomers, while AHH activity, representative of 3-MC type induction, was increased by other PCB isomers. The absorption spectrum of the cytochrome P-450

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induced by the latter isomers was similar to that found with 3-MC induction, which causes a peak absorption at 448 nm, not 450 nm, and is thus referred to as cytochrome P-448. The absorption spectrum of the cytochrome P-450 induced by isomers with PB-type activity was similar to the PB-induced cytochrome P-450 spectrum with a peak absorption at 450 nm.

The toxicity of the individual isomers was also correlated to their type of MFO induction. The 3-MC type inducers were more toxic than PB type inducers. This toxicity, however, was not directly related to the induction of cytochrome P-448/P-450 (Yoshimura et al. 1979).

McKinney and Singh (1981) examined crystallographic, theoretical and toxicological data in developing an hypothesis on the structural specificity of PCB for 3-MC-type induction of cytochrome P-448. They found that the planar structure of PCBs with chlorines at the para positions of each ring and at least one meta position of each ring was very similar to the planar structure of 2,3,7,8-tetrachlorodibenzo-p-dioxin, a very potent 3-MC-type inducer. Such a structure can fit into a 3 x 10 Å rectangle. In addition to the ability to form this planar structure, relatively high polarizability of the molecule, which is determined by the number and position of the chlorines, is important for the molecule to have a high affinity to the receptor site responsible for MFO-induction.

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## 7. Induction of Porphyria

Porphyria cutanea tarda in humans is an acquired defect in hepatic porphyrin metabolism, characterized by uroporphorinuria (excretion of porphyrins in the urine), photosensitivity as manifested by blisters, and mechanical fragility of the skin (Kimbrough 1974). Normally, porphyrin synthesis is well regulated to provide the necessary amount of porphyrins for heme synthesis. The hepatic porphyria which is responsible for an increase in porphyrins and for the skin photosensitivity can be produced experimentally by a number of drugs and chemicals which have the ability to stimulate activity of the initial enzyme of heme synthesis,  $\delta$ -aminolevulinic acid (ALA)-synthetase (Strik and Wit 1972).

PCBs have been shown to cause hepatic porphyria in animals (Goldstein et al. 1974, Vos and Beems 1971). Goldstein et al. (1974) fed rats a diet containing 100 ppm Aroclor 1254 for up to 13 months. After 2-7 months, there was an increase in the urinary excretion of several intermediary compounds of the metabolic pathway for heme synthesis. Uroporphyrin excretion increased up to 540-fold while the excretion of coproporphyrin increased 27-fold, porphobilinogen increased 50-fold, and  $\delta$ -ALA increased 18-fold. The activity of  $\delta$ -ALA synthetase, the rate-limiting enzyme of the metabolic pathway, was increased after 3 months on the diet. At 4 months, the porphyrin concentration in the livers of treated rats was 1410  $\mu\text{g/g}$  of liver compared to the concentration in the livers of control animals

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of  $<0.1 \mu\text{g/g}$ . Aroclors 1016 and 1242 also induce porphyria in rats (Iverson et al. 1975, Goldstein et al. 1975). After dosing at 10 and 100 mg/kg/day for 21 days, both mixtures caused a significant accumulation of porphyrins in both sexes, but Aroclor 1242 was nearly twice as effective at 100 mg/kg and produced a significant effect at the lower dose.

Rabbits developed symptoms of porphyria after application of Clophen A50, Phenoclor DP6, and Aroclor 1260 to their back skin (Vos and Beems 1971).

## 8. Immunotoxicity

The ability of PCBs to inhibit immune responses has been reviewed by Vos (1977) and by Silkworth and Loose (1981). Treatment with PCBs has been shown to reduce the level of serum antibodies to pseudorabies virus in rabbits and to tetanus toxin in guinea pigs, reduce the delayed-type hypersensitivity response to tuberculin in rabbits and to Freund's complete adjuvant in guinea pigs, reduce the mixed lymphocyte response in guinea pigs, and increase the susceptibility of ducks to infection with duck hepatitis virus. PCBs also cause atrophy of the thymus and damage to the spleen in several species. Since the thymus and spleen are involved in immunologic reactions, these adverse effects are indicators of reduced immunocompetence.

More recently, Aroclor 1254 at 63, 135, 250 and 550 mg/kg has been shown to produce a dose-dependent reduction in plaque-forming cells in the spleen of mice in response to a challenge

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with sheep red blood cells. This indicates that Aroclor 1254 inhibited the formation of antibody-producing cells in the spleen (Wierda et al. 1981). A similar effect was seen in Rhesus monkeys fed Aroclor 1248 at 5 ppm in the diet for 11 months. These animals had reduced hemolysin titers in response to injection with washed sheep red blood cells and reduced levels of  $\gamma$ -globulin in blood serum compared to control animals not exposed to PCBs (Thomas and Hinsdill 1978).

## C. Toxicity of Chlorinated Dibenzofurans (CDFs)

As described earlier, commercial PCBs are contaminated with low levels of CDFs. CDFs may be formed from PCBs during heating and during use in heat exchangers, and high levels of CDFs were found in the rice oil responsible for the Yusho incident in Japan. CDFs have been detected in the adipose tissue and liver of Yusho victims at levels of about 0.01 ppm on a whole tissue basis (Kuratsune et al. 1976). Indeed they appear to be preferentially retained compared to PCBs since the ratio of PCB to CDF in the livers of the patients 7 years after exposure was about 4:1 compared to 200:1 in the contaminated rice oil.

The association of CDFs with PCBs and the apparent formation of CDFs from PCBs when heated are of particular concern because CDFs are considerably more toxic than PCBs. For example, in a comparative toxicity study, Oishi et al. (1978) examined the toxicity of a PCB mixture (Kanechlor 500) and a mixture of CDFs produced by chlorination of dibenzofuran. The CDF

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mixture contained tetrachloro-, pentachloro-, and hexachloro-dibenzofurans (average number of chlorines per molecule, 4.7). Groups of 10 male Sprague-Dawley rats were fed for 4 weeks on diets containing 100 ppm PCB, 10 ppm CDF, or 1 ppm CDF; an additional group was given untreated feed. Body weight gains were less in the treated groups than in the control group. This effect increased in severity in the order 100 ppm PCB < 1 ppm CDF < 10 ppm CDF. The relative weights (g/100 g body weight) of brain, spleen, and liver were significantly increased in the animals fed 100 ppm PCBs. These effects were also produced by both dose levels of CDFs. In addition, both 1 ppm and 10 ppm CDFs significantly increased the relative weights of heart, lungs, adrenals, and testes, and significantly decreased the absolute and relative thymus weight. CDFs at 10 ppm also significantly decreased the relative weights of seminal vesicles and ventral prostate. Both doses of CDFs significantly reduced the blood hemoglobin concentration and hematocrit compared to the control group. This effect was not produced by the PCB treatment. All three treatments significantly increased the serum cholesterol concentration, significantly reduced the serum triglyceride concentration and significantly increased the concentration of lipid in the liver. It appears that 1 ppm CDF produced effects more severe than 100 ppm PCB in this study.

Although the toxicity of individual isomers of CDFs has not been studied in detail, emphasis has been placed on the isomer 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) since

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it is likely to be the most toxic by analogy with the structurally similar chlorodibenzo-p-dioxins (CDD), of which 2,3,7,8-TCDD is the most toxic (Poland and Glover 1973). 2,3,7,8-TCDF is also one of the isomers that is found in commercial PCBs (Bowes et al. 1975a, b), is formed during heating of PCBs (Morita et al. 1978), and is the major CDF in Yusho oil, in which it was present at 0.45 ppm, about 8% of the total CDFs (Buser et al. 1978a).

In rhesus monkeys, 2,3,7,8-TCDF at 5 and 50 ppb in the diet caused sickness and some deaths in groups of three animals fed for 6 and 2 months, respectively. The principal pathologic effects were atrophy or squamous metaplasia of the sebaceous glands, mucous metaplasia and hyperplasia of the gastric mucosa, involution of the thymus, and hypoplasia of the bone marrow. The effects were most severe at the 50 ppm dose level, but were also apparent at the lower dose level. The survivors recovered after 3 months on a TCDF-free diet (McNulty et al. 1981)

CDFs are also much more potent inducers of liver microsomal enzymes than PCBs. Kawano and Hiraga (1978) observed a greater increase in liver cytochrome P-450 content in rats given three daily doses of mixed CDFs at 100 µg/kg/day than in rats given three daily doses of a PCB mixture (Kanechlor 500) at 10 mg/kg/day. Also, Goldstein et al. (1978) observed that the highly toxic isomer 2,3,7,8-TCDF was active as an inducer of the liver microsome enzyme aryl hydrocarbon hydroxylase in rats when given

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as three daily intraperitoneal doses of 0.1 µg/kg/day. This was the lowest dose tested, and a no-effect level for this isomer has not been identified. Indeed, contamination of a commercial, 99% pure hexachlorobiphenyl isomer with 44 ppm 2,3,7,8-TCDF was responsible for the induction of cytochrome P-448 and aryl hydrocarbon hydroxylase by the commercial hexachlorobiphenyl (Goldstein et al. 1978). These results indicate that 2,3,7,8-TCDF was at least 20,000 times more potent as an inducer of AHH than 2,2',4,4',5,5'-hexachlorobiphenyl.



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## APPENDIX E

### TOXICOLOGICAL DATA NOT USED IN THE RISK ASSESSMENT

In the text of this report, we selected three types of toxic effect of PCBs as the basis for assessment of risks at low exposure levels. Chloracne is the best documented effect in humans and is an effect for which the exposure-response relationship has been defined reasonably clearly. Reproductive toxicity is the effect that has been observed in animal experiments at the lowest dose level. Carcinogenesis in animals is an effect of significant public health concern and an effect for which there are scientific reasons to believe that risks may exist at very low dose levels.

In addition to these types of toxic effect, several other effects of PCBs have been reported, but were not used in our risk assessment. The most important of these reported effects are listed below.

1. Data suggesting abnormal lung function in workers exposed to PCBs (Warshaw et al. 1979). In our judgment these findings need independent confirmation.

2. Data suggesting correlations between PCB levels in tissues and indicators of cardiovascular risks (Kreiss et al. 1981, Baker et al. 1980). Although these are potentially significant risk factors, it is not clear whether the elevated PCB levels associated with them are causes or effects.

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3. Data on mutagenicity of lower chlorinated biphenyls (Wyndham et al. 1976) and on binding of metabolites to DNA and RNA (Morales and Matthews 1979). The significance of these findings for risk assessment is not clear, since the mutagenic components are not found in commercial mixtures such as Aroclor 1254.

4. Data on the sensitivity of mink to low levels of PCBs. The validity of the mink as an experimental model for toxic effects in humans is questionable.

5. Data on teratogenic effects of PCBs. Studies by Earl et al. (1974) have not been reported in sufficient detail; Lucier et al. (1978) used 3,3',4,4'-tetrachlorobiphenyl, a highly toxic isomer that is only a minor component in commercial mixtures.

6. Data on induction of liver enzymes at low doses. Although several human studies have shown increased activity of certain liver enzymes (SGOT, SGPT, and GGP), the health significance of these findings is not clear. In theory, the increased activity of mixed function oxidases could either increase the risks of cancer (by augmenting the conversion of carcinogens into active metabolites), or decrease it (by accelerating the breakdown of these active metabolites). In practice, most studies have indicated a decrease in effects when PCBs are administered prior to administration of a carcinogen that requires metabolic activation (see Appendix D).

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Hence, we will assume that the net effect of enzyme induction by PCBs is likely to be beneficial or neutral.

In addition to toxic effects of PCBs themselves, it is also necessary to consider possible risks posed by the toxic contaminants that can be formed from PCBs, primarily the chlorinated dibenzofurans (CDFs). Toxicity studies of CDFs are limited, but chronic studies in monkeys suggest that CDFs are on the order of 1,000 times more toxic than PCBs tested under similar conditions (McNulty et al. 1981, Allen et al. 1979). This is consistent with data from the "Yusho" poisoning incident in Japan, in which the lowest daily doses ingested by individuals who showed clear signs of poisoning were about 0.16 µg/kg/day of CDFs (Hayabachi et al. 1979). This suggests that the toxicity of CDFs would not need to be considered unless they occurred with PCBs in a ratio greater than about 1:1,000. Testing at Minerva indicated that CDFs occur in the environmental residues of PCBs in a ratio of only about 1:1,000,000. This ratio is similar to that found in the original commercial mixture, Aroclor 1254 (Bowes et al. 1975). Hence the toxicity of the environmental residues should be adequately characterized by the results of studies with the commercial mixture, and it is not necessary to consider the effects of CDFs separately in the risk assessment.

# TRW PROPRIETARY INFORMATION

## REFERENCES (Appendixes E and F)

- ALLEN, J., and NORBACK, D. 1976. Pathobiological responses of primates to polychlorinated biphenyl exposure. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 57-64
- ALLEN, J., BARSOTTI, D., LAMBRECHT, L., and VAN MILLER, J. 1979. Reproductive effects of halogenated aromatic hydrocarbons on nonhuman primates. Ann. NY Acad. Sci. 320:419-425
- ALLEN, J., BARSOTTI, D., and CARSTENS, L., 1980. Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. J. Toxicol. Environ. Health 6:55-66
- ALVARES, A.P., BICKERS, D.R., and KAPPAS, A. 1973. Polychlorinated biphenyls: A new type of inducer of cytochrome P-448 in the liver. Proc. Nat. Acad. Sci. USA 70:1321-1325
- ALVARES, A.P., FISHBEIN, A., ANDERSON, K.E., and KAPPAS, A. 1977. Alterations in drug metabolism in workers exposed to polychlorinated biphenyls. Clin. Pharmacol. Ther. 22:140-146
- AULERICH, R., and RINGER, R. 1977. Current status of PCB toxicity to mink and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279-92
- AULERICH, R., and RINGER, R. 1980. Toxicity of the polychlorinated biphenyl Aroclor 1016 to mink. U.S. Environmental Protection Agency. EPA-600/3-80-033 (PB80-16853-7)
- BAHN, A.K., ROSENWAIKE, I., HERRMANN, N., GROVER, P., STELLMAN, J., and O'LEARY, K. 1976. Melanoma after exposure to PCB's. N. Engl. J. Med. 295:450
- BAKER, E.L., Jr., LANDRIGAN, P.J., GLUECK, C.J., ZACK, M.M., Jr., LIDDLE, J.A., BURSE, V.W., HOUSWORTH, W.J., and NEEDHAM, L.L. 1980. Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. Am. J. Epidemiol. 112:553-563
- BARSOTTI, D.A., and ALLEN, J.R. 1975. Effects of polychlorinated biphenyls on reproduction in the primate. Fed. Proc. 34:338

# TRW PROPRIETARY INFORMATION

## REFERENCES (Appendixes D and F)

- ALLEN, J., and NORBACK, D. 1976. Pathobiological responses of primates to polychlorinated biphenyl exposure. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 57-64
- ALLEN, J., BARSOTTI, D., LAMBRECHT, L., and VAN MILLER, J. 1979. Reproductive effects of halogenated aromatic hydrocarbons on nonhuman primates. Ann. NY Acad. Sci. 320:419-425
- ALLEN, J., BARSOTTI, D., and CARSTENS, L., 1980. Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. J. Toxicol. Environ. Health 6:55-66
- ALVARES, A.P., BICKERS, D.R., and KAPPAS, A. 1973. Polychlorinated biphenyls: A new type of inducer of cytochrome P-448 in the liver. Proc. Nat. Acad. Sci. USA 70:1321-1325
- ALVARES, A.P., FISHBEIN, A., ANDERSON, K.E., and KAPPAS, A. 1977. Alterations in drug metabolism in workers exposed to polychlorinated biphenyls. Clin. Pharmacol. Ther. 22:140-146
- AULERICH, R., and RINGER, R. 1977. Current status of PCB toxicity to mink and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279-92
- AULERICH, R., and RINGER, R. 1980. Toxicity of the polychlorinated biphenyl Aroclor 1016 to mink. U.S. Environmental Protection Agency. EPA-600/3-80-033 (PB80-16853-7)
- BAHN, A.K., ROSENWAIKE, I., HERRMANN, N., GROVER, P., STELLMAN, J., and O'LEARY, K. 1976. Melanoma after exposure to PCB's. N. Engl. J. Med. 295:450
- BAKER, E.L., Jr., LANDRIGAN, P.J., GLUECK, C.J., ZACK, M.M., Jr., LIDDLE, J.A., BURSE, V.W., HOUSWORTH, W.J., and NEEDHAM, L.L. 1980. Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. Am. J. Epidemiol. 112:553-563
- BARSOTTI, D.A., and ALLEN, J.R. 1975. Effects of polychlorinated biphenyls on reproduction in the primate. Fed. Proc. 34:338

# TRW PROPRIETARY INFORMATION

- BERRY, D.L., DIGIOVANNI, J., JUCHAU, M.R., BRACKEN, W.H., GLEASON, G.L., and SLAGA, T.J. 1978. Lack of tumor-promoting ability of certain environmental chemicals in a two-stage mouse skin tumorigenesis assay. Res. Commun. Chem. Pathol. Pharmacol. 20:101-108
- BITMAN, J., CECIL, H.C., and HARRIS, S.J. 1972. Biological effects of polychlorinated biphenyls in rats and quail. Environ. Health Perspect. 1:145-149
- BLEAVINS, M.R., AULERICH, R.H., and RINGER, R.K. 1980. Polychlorinated biphenyls (Aroclors 1016 and 1242): Effects on survival and reproduction in mink and ferrets. Arch. Environ. Contam. Toxicol. 9:627-635
- BOWES, G.W., MULVIHILL, M.J., SIMONEIT, B.R.T., BURLINGAME, A.L., and RISEBROUGH, R.W. 1975a. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 256:305-307
- BOWES, G.W., MULVIHILL, M.J., DECAMP, M.R., and KENDE, A.S. 1975b. Gas chromatographic characteristics of authentic chlorinated dibenzofurans; identification of two isomers in American and Japanese polychlorinated biphenyls. J. Agric. Food Chem. 23:1222-1223
- BOWMAN, R., HEIRONIMUS, M., and ALLEN, J. 1978. Correlations of PCB body burden with behavioral toxicology in monkeys. Pharmacol. Biochem. Behav. 9:49-56
- BOWMAN, R., HEIRONIMUS, M., and BARSOTTI, D. 1981. Locomotor hyperactivity in PCB-exposed Rhesus monkeys. Neurotoxicology 2: 251-268
- BROWN, D.P., and JONES, M. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch. Environ. Health 36:120-129
- BURSE, V.W., KIMBROUGH, R.D., VILLANUEVA, E.C., JENNINGS, R.W., LINDER, R.E., and SOROCOOL, G.W. 1974. Polychlorinated biphenyls. Storage, distribution, excretion, and recovery: Liver morphology after prolonged dietary ingestion. Arch. Environ. Health 29:301-307
- BUSER, H.R., RAPPE, C., and GARA, A. 1978a. Polychlorinated dibenzofurans (PCDFs) found in Yusho oil and in used Japanese PCB. Chemosphere 7:439-449
- BUSER, H.R., BOSSHARDT, H.P., and RAPPE, C. 1978b. Formation of polychlorinated dibenzofurans (PCDFs) from the pyrolysis of PCBs. Chemosphere 7:109-119

# TRW PROPRIETARY INFORMATION

- CHANG, K.J., CHENG, J.S., HUANG, P.C., and TUNG, T.C. 1980a. [Study of patients with PCB poisoning.] J. Formosan Med. Assoc. 79:304-313
- CHANG, K.J., LU, F.J., TUNG, T.C., and LEE, T.P. 1980b. Studies on patients with PCB poisoning. 2. Determination of urinary coproporphyrin, uroporphyrin, w-aminolevulinic acid and porphobilinogen. Res. Commun. Chem. Pathol. Pharmacol. 30:547-554
- CHANG, K.J., HSIEH, K.H., LEE, T.P., TANG, S.Y., and TUNG, T.C. 1981. Immunologic evaluation of patients with polychlorinated biphenyl poisoning: Determination of lymphocyte subpopulations. Toxicol. Appl. Pharmacol. 61:58-63
- CHEN, T.S., and DUBOIS, K.P. 1973. Studies on the enzyme inducing effect of polychlorinated biphenyls. Toxicol. Appl. Pharmacol. 26:504-512
- CRUMP, K.S., and MASTERMAN, M.D. 1979. Assessment of Carcinogenic Risks from PCBs in Food. Unpublished Report to U.S. Congress, Office of Technology Assessment. April 1979
- EARL, F., COUVILLION, J. and Van LOON, E. 1974. The reproductive effects of PCB 1254 in beagle dogs and miniature swine. Toxicol. Appl. Pharmacol. 29:104 (Abstract)
- ECOBICHON, D.J. 1976. Enzymatic and other biochemical responses to selected PCB's. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 75-87
- FISCHBEIN, A., WOLFF, M.S., LILIS, R., THORNTON, J., and SELIKOFF, I.J. 1979. Clinical findings among PCB-exposed capacitor manufacturing workers. Ann. NY Acad. Sci. 320:703-715
- GOLDSTEIN, J.A. 1979. The structure-activity relationships of halogenated biphenyls as enzyme inducers. Ann. NY Acad. Sci. 320:164-178
- GOLDSTEIN, J.A., HICKMAN, P., and JUE, D.L. 1974. Experimental hepatic porphyria induced by polychlorinated biphenyls. Toxicol. Appl. Pharmacol. 27:437-448
- GOLDSTEIN, J.A., HICKMAN, P., BURSE, V.W., and BERGMAN, H. 1975. A comparative study of two polychlorinated biphenyl mixtures (Aroclors 1242 and 1016) containing 42% chlorine on induction of hepatic porphyria and drug metabolizing enzymes. Toxicol. Appl. Pharmacol. 32:461-473

# TRW PROPRIETARY INFORMATION

- GOLDSTEIN, J.A., HICKMAN, P., BERGMAN, H., McKINNEY, J.D., and WALKER, M.P. 1977. Separation of pure polychlorinated biphenyl isomers into two types of inducers on the basis of induction of cytochrome P-450 or P-448. Chem.-Biol. Interact. 17:69-87
- GOLDSTEIN, J.A., HASS, J.R., LINKO, P., and HARVAN, D.J. 1978. 2,3,7,8-Tetrachlorodibenzofuran in a commercially available 99% pure polychlorinated biphenyl isomer identified as the inducer of hepatic cytochrome P-448 and aryl hydrocarbon hydroxylase in the rat. Drug Metab. Dispos. 6:258-264
- GREEN, S., CARR, J.V., PALMER, K.A., and OSWALD, E.J. 1975a. Lack of cytogenetic effects in bone marrow and spermatogonial cells in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). Bull. Environ. Contam. Toxicol. 13:14-22
- GREEN, S., SAURO, F.M., and FRIEDMAN, L. 1975b. Lack of dominant lethality in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). Food Cosmet. Toxicol. 13:507-510
- HAMMOND, P.B., NISBET, I.C.T., and SAFOFIM, A.F. 1972. Polychlorinated biphenyls: Environmental impact. Environ. Res. 5:249-362
- HAYABUCHI, H., YOSHIMURA, T., and KURATSUNE, M. 1979. Consumption of toxic rice oil by 'Yusho' patients and relation to the clinical response and latent period. Food Cosmet. Toxicol. 17:455-461
- HSIA, M.T.S., LIN, F.S.D., and ALLEN, J.R. 1978. Comparative mutagenicity and toxic effects of 2,5,2',5'-tetrachlorobiphenyl and its metabolites in bacterial and mammalian test systems. Res. Commun. Chem. Pathol. Pharmacol. 21:485-496
- HUMPHREY, H.E.B. 1977. Evaluation of Changes of the Level of Polychlorinated Biphenyls (PCB) in Human Tissue. Final Report on FDA Contract 223-73-2209
- HUTZINGER, O., SAFE, S., and ZITKO, V. 1974. The Chemistry of PCB's. CRC Press, Cleveland, Ohio. 77 pp.
- ITO, N., TATEMATSU, M., HIROSE, M., NAKANISHI, K., and MURASAKI, G. 1978. Enhancing effect of chemicals on production of hyperplastic liver nodules induced by N-2-fluorenylacetamide in hepatectomized rats. Gann 69:143-144
- ITO, Y., UZAWA, H., and NOTOMI, A. 1971. Lipid composition of the rabbit liver poisoned with chlorobiphenyls. Fukuoka Acta Medica 62:48-50



# TRW PROPRIETARY INFORMATION

- IVERSON, F., VILLENEUVE, D.C., GRANT, D.L., and HATINA, G.V. 1975. Effect of Aroclor 1016 and 1242 on selected enzyme systems in the rat. Bull. Environ. Contam. Toxicol. 13:456-463
- KAPPAS, A., and ALVARES, A.P. 1975. How the liver metabolizes foreign substances. Scientific American 232:22-31
- KAWANO, S., and HIRAGA, K. 1978. Polychlorinated dibenzofurans--potent inducers of rat hepatic drug metabolizing enzymes. Japan. J. Pharmacol. 28:305-315
- KIMBROUGH, R.D. 1974. The toxicity of polychlorinated polycyclic compounds and related chemicals. CRC Crit. Rev. Toxicol. 2:445-498
- KIMBROUGH, R.D., and LINDER, R.E. 1974. Induction of adenofibrosis and hepatomas of the liver in BALB/cJ mice by polychlorinated biphenyls (Aroclor 1254). JNCI 53:547-552
- KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J., and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. JNCI 55:1453-1456
- KIMBROUGH, R., BUCKLEY, J., FISHBEIN, L., FLAMM, G., KASZA, L., MARCUS, W., SHIBKO, S., and TESKE, R. 1978. Animal toxicology. Environ. Health Perspect. 24:173-184
- KIMURA, N.T., KANEMATSU, T., and BABA, T. 1976. Polychlorinated biphenyl(s) as a promoter in experimental hepatocarcinogenesis in rats. Z. Krebsforsch. 87:257-266
- KREISS, K., ZACK, M.M., KIMBROUGH, R.D., NEEDHAM, L.L., SMREK, A.L., and JONES, B.T. 1981. Association of blood pressure and polychlorinated biphenyl levels. JAMA 245:2505-2509
- KUWABARA, K., YAKUSHIJI, T., WATANABE, I., YOSHIDA, S., KOYAMA, K., KUNITA, N., and HARA, I. 1978. Relationship between breast feeding and PCB residues in blood of the children whose mothers were occupationally exposed to PCBs. Int. Arch. Occup. Environ. Health 41:189-197
- LINDER, R.E., GAINES, T.B., and KIMBROUGH, R.D. 1974. The effect of polychlorinated biphenyls on rat reproduction. Food Cosmet. Toxicol. 12:63-77
- LITTERST, C.L., FARBER, T.M., BAKER, A.M., and VAN LOON, E.J. 1972. Effect of polychlorinated biphenyls on hepatic microsomal enzymes in the rat. Toxicol. Appl. Pharmacol. 23:112-122

# TRW PROPRIETARY INFORMATION

- LITTERST, C.L., and VAN LOON, E.J. 1974. Time-course of induction of microsomal enzymes following treatment with polychlorinated biphenyl. Bull. Environ. Contam. Toxicol. 11: 206-212
- LUCIER, G., DAVIS, G., and McLACHLAN, J. 1978. Transplacental toxicology of the polychlorinated and polybrominated biphenyls. Dept. of Energy Symposia Series Vol. 47: Toxicology of Energy-Related Pollutants. Pp. 188-203
- MAKIURA, D., AOE, H., SUGIHARA, S., HIRAO, K., ARAI, M., and ITO, N. 1974. Inhibitory effects of polychlorinated biphenyls on liver tumorigenesis in rats treated with 3'-methyl-4-dimethylaminoazobenzene, N-2-fluorenylacetamide, and diethylnitrosamine. JNCI 53:1253-1257
- MARKS, T., KIMMEL, G., and STAPLES, R. 1981. Influence of symmetrical polychlorinated biphenyl isomers on embryo and fetal development in mice. I. Teratogenicity of 3,3',-4,4',5,5'-hexachlorobiphenyl. Toxicol. Appl. Pharmacol. 61:269-276
- McKINNEY, J.D., and SINGH, P. 1981. Structure-activity relationships in halogenated biphenyls: Unifying hypothesis for structural specificity. Chem. Biol. Interact. 33:271-284
- McMAHON, R.E., CLINE, J.C., and THOMPSON, C.Z. 1979. Assay of 855 test chemicals in ten tester strains using a new modification of the Ames test for bacterial mutagens. Cancer Res. 39:682-692
- McNULTY, W.P. 1976. Use of the rhesus monkey as a model for human toxicity with polychlorinated biphenyls. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 332-333
- McNULTY, W.P., POMERANTZ, I., and FARRELL, T. 1981. Chronic toxicity of 2,3,7,8-tetrachlorodibenzofuran for Rhesus macaques. Food Cosmet. Toxicol. 19:57-65
- MIYATA, H., MURAKAMI, Y., and KASHIMOTO, T. 1978. Studies on the compounds related to PCB (VI). Determination of polychlorinated quaterphenyl (PCQ) in Kanemi rice oil caused the 'Yusho' and investigation on the PCQ formation. J. Food Hyg. Soc. Japan 19:417-425
- MIYATA, H., MURAKAMI, Y., KASHIMOTO, T., and KUNITA, N. 1979. [Organochlorine compounds in contaminated rice oils.] Osaka-furitsu Koshu Eisei Kenkyusho Konkyu Hokoku, Shokuhin Eisei Hen 9:95-103. As cited in Chem. Abst. 92:1187C

# TRW PROPRIETARY INFORMATION

- MOORE, J.A., GUPTA, B.N., and VOS, J.G. 1976. Toxicity of 2,3,7,8-tetrachlorodibenzofuran - Preliminary results. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 77-80
- MORITA, M., NAKAGAWA, J., AKIYAMA, K., MIMURA, S., and ISONO, N. 1977. Detailed examination of polychlorinated dibenzofurans in PCB preparations and Kanemi Yusho oil. Bull. Environ. Contam. Toxicol. 18:67-73
- MORITA, M., NAKAGAWA, J., and RAPPE, C. 1978. Polychlorinated dibenzofuran (PCDF) formation from PCB mixture by heat and oxygen. Bull. Environ. Contam. Toxicol. 19:665-670
- MURAI, Y., and KUROIWA, Y. 1971. Peripheral neuropathy in chlorobiphenyl poisoning. Neurol. 21:1173-1176
- NAGAYAMA, J., KURATSUNE, M., and MASUDA, Y. 1976. Determination of chlorinated dibenzofurans in Kanechlors and "Yusho oil." Bull. Environ. Contam. Toxicol. 15:9-13
- NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. NCI Carcinogenesis Technical Report No. 38
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Polychlorinated Biphenyls (PCBs). DHEW Publication No. (NIOSH) 77-225
- NILSSON, B., and RAMEL, C. 1974. Genetic tests on Drosophila melanogaster with polychlorinated biphenyls (PCB). Hereditas 77:319-322
- NISBET, I.C.T. 1976. Environmental transport and occurrence of PCBs in 1975. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 245-247
- NISBET, I.C.T. 1981. Health Risks Posed to Consumers of Fish Contaminated with PCBs from Lake Michigan. Report Prepared for U.S. Environmental Protection Agency, Region V, Chicago, Illinois. February 1981
- NISHIZUMI, M. 1976. Enhancement of diethylnitrosamine hepatocarcinogenesis in rats by exposure to polychlorinated biphenyls or phenobarbital. Cancer Lett. 2:11-16
- NISHIZUMI, M. 1979. Effect of phenobarbital, dichlorodiphenyl-trichloroethane and polychlorinated biphenyls on diethylnitrosamine-induced hepatocarcinogenesis. Gann 70:835-837

# TRW PROPRIETARY INFORMATION

- NISHIZUMI, M. 1980. Reduction of diethylnitrosamine induced hepatoma in rats exposed to polychlorinated biphenyls through their dams. *Gann* 71:910-912
- NORBACK, D.H., and ALLEN, J.R. 1972. Chlorinated aromatic hydrocarbon induced modifications of the hepatic endoplasmic reticulum: Concentric membrane arrays. *Environ. Health Perspect.* 1:137-143
- OISHI, S., MORITA, M., and FUKUDA, H. 1978. Comparative toxicity of polychlorinated biphenyls and dibenzofurans in rats. *Toxicol. Appl. Pharmacol.* 43:13-22
- PITOT, H.C., and SIRICA, A.E. 1980. The stages of initiation and promotion in hepatocarcinogenesis. *Biochim. Biophys. Acta* 605:191-215
- POLAND, A., and GLOVER, E. 1973. Studies on the mechanism of toxicity of the chlorinated dibenzo-p-dioxins. *Environ. Health Perspect.* 5:245-251
- POLAND, A., and GLOVER, E. 1977. Chlorinated biphenyl induction of aryl hydrocarbon hydroxylase activity: A study of the structure-activity relationship. *Molec. Pharmacol.* 13:924-938
- PRESTON, B.D., VAN MILLER, J.P., MOORE, R.W., and ALLEN, J.R. 1981. Promoting effects of polychlorinated biphenyls (Aroclor 1254) and polychlorinated dibenzofuran-free Aroclor 1254 on diethylnitrosamine-induced tumorigenesis in the rat. *JNCI* 66:509-515
- RYAN, D.E., THOMAS, P.E., KORZENIOWSKI, D., and LEVIN, W. 1979. Separation and characterization of highly purified forms of liver microsomal cytochrome P-450 from rats treated with polychlorinated biphenyls, phenobarbital, and 3-methylcholanthrene. *J. Biol. Chem.* 254:1365-1374
- SCHOENY, R.S., SMITH, C.C., and LOPER, J.C. 1979. Non-mutagenicity for *Salmonella* of the chlorinated hydrocarbons Aroclor 1254, 1,2,4-trichlorobenzene, mirex and kepone. *Mutat. Res.* 68:125-132
- SHIGEMATSU, N., ISHIMARU, S., SAITO, R., IKEDA, T., MATSUBA, K., SUGIYAMA, K., and MASUDA, Y. 1978. Respiratory involvement in polychlorinated biphenyls poisoning. *Environ. Res.* 16:92-100
- SILKWORTH, J.B., and LOOSE, L.D. 1981. Assessment of the environmental contaminant-induced lymphocyte dysfunction. *Environ. Health Perspect.* 39:105-128

# TRW PROPRIETARY INFORMATION

- STRIK, J.J.T.W.A., and WIT, J.G. 1972. Hepatic porphyria in birds and mammals. TNO-Nieuws 27:604-610
- TATEMATSU, M., NAKANISHI, K., MURASAKI, G., MIYATA, Y., HIROSE, M., and ITO, N. 1979. Enhancing effect of inducers of liver microsomal enzymes on induction of hyperplastic liver nodules by N-2-fluorenylacetamide in rats. JNCI 63:1411-1416
- THOMAS, P.T., and HINS DILL, R.D. 1978. Effect of polychlorinated biphenyls on the immune responses of Rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44:41-51
- TILSON, H.A., DAVIS, G.J., McLACHLAN, J.A., and LUCIER, G.W. 1979. The effects of polychlorinated biphenyls given prenatally on the neurobehavioral development of mice. Environ. Res. 18:466-474
- UCHIYAMA, M., CHIBA, T., and NODA, K. 1974. Co-carcinogenic effect of DDT and PCB feedings on methylcholanthrene-induced chemical carcinogenesis. Bull. Environ. Contam. Toxicol. 12:687-693
- UNGER, M., and OLSEN, J. 1980. Organochlorine compounds in the adipose tissue of deceased people with and without cancer. Environ. Res. 23:257-263
- URABE, H., KODA, H., and ASAHI, M. 1979. Present state of Yusho patients. Ann. NY Acad. Sci. 320:273-276
- VILLENEUVE, D.C., GRANT, D.L., KHERA, K., CLEGG, D.J., BAER, H., and PHILLIPS, W.E.J. 1971. The fetotoxicity of a polychlorinated biphenyl mixture (Aroclor 1254) in the rabbit and in the rat. Environ. Physiol. 1:67-71
- VOS, J.G. 1977. Immune suppression as related to toxicology. CRC Crit. Rev. Toxicol. 5:67-101
- VOS, J.G., and BEEMS, R.B. 1971. Dermal toxicity studies of technical polychlorinated biphenyls and fractions thereof in rabbits. Toxicol. Appl. Pharmacol. 19:617-633
- WARSHAW, R., FISCHBEIN, A., THORNTON, J., MILLER, A., and SELIKOFF, I.J. 1979. Decrease in vital capacity in PCB-exposed workers in a capacitor manufacturing facility. Ann. NY Acad. Sci. 320:277-283
- WASSERMANN, M., NOGUEIRA, D.P., CUCOS, S., MIRRA, A.P., SHIBATA, H., ARIE, G., MILLER, H., and WASSERMANN, D. 1978. Organochlorine compounds in neoplastic and adjacent apparently normal gastric mucosa. Bull. Environ. Contam. Toxicol. 20:544-553

# TRW PROPRIETARY INFORMATION

- WATANABE, M., and SUGAHARA, T. 1981. Experimental formation of cleft palate in mice with polychlorinated biphenyls. Toxicology 19:49-54
- WEBB, R.G., and McCALL, A.C. 1973. Quantitative PCB standards for electron capture gas chromatography. J. Chromatographic Sci. 11:366-373
- WIERDA, D., IRONS, R.D., and GREENLEE, W.F. 1981. Immunotoxicity in C57BL/6 mice exposed to benzene and Aroclor 1254. Toxicol. Appl. Pharmacol. 60:410-417
- WORLD HEALTH ORGANIZATION (WHO). 1978. Environmental Health Criteria for Polychlorinated Biphenyls and Polychlorinated Terphenyls. WHO, Geneva, Switzerland
- WYNDHAM, C., DEVENISH, J., and SAFE, S. 1976. The in vitro metabolism, macromolecular binding and bacterial mutagenicity of 4-chlorobiphenyl, a model PCB substrate. Res. Commun. Chem. Pathol. Pharmacol. 15:563-570
- WYNDHAM, C., and SAFE, S. 1978. In vitro metabolism of 4-chlorobiphenyl by control and induced rat liver microsomes. Biochemistry 17:208-215
- YOSHIMURA, H., YOSHIHARA, S., OZAWA, N., and MIKI, M. 1979. Possible correlation between induction modes of hepatic enzymes by PCBs and their toxicity in rats. Ann. NY Acad. Sci. 320:179-192



**CHARACTERIZATION, RISK ASSESSMENT  
AND REMEDIAL ACTION PLAN  
FOR A PCB SPILL AT  
THE TRW SITE IN MINERVA, OHIO**

**Volume 1**

118499

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## EXECUTIVE SUMMARY

In August 1981, TRW notified the U.S. Environmental Protection Agency (EPA) and the Ohio Environmental Protection Agency (Ohio EPA) of its discovery of polychlorinated biphenyls (PCBs) in the soil at the southeast corner of their plant in Minerva, Ohio. Plant records suggested that spent oil used in diffusion pumps that contained PCB (principally Aroclor 1254) had been stored in this area. This discovery warranted further investigation.

With the concurrence of EPA and Ohio EPA, TRW proceeded to investigate and rectify the situation. Because use of such oils was discontinued in 1973, the source of PCBs had been removed from the site. Remaining signs of past spills, such as drum debris and oil stains, were also removed from the site. Limited soil and water sampling by TRW, EPA, and Ohio EPA indicated that residual PCBs were present in soils and sediments on several areas of the site. Since residual PCB concentrations at the site did not pose an imminent hazard to the surrounding population, TRW undertook to study the situation in detail and develop appropriate measures for mitigating potential long-term effects.

In October 1981, TRW retained Clement Associates to perform a thorough assessment of the extent and distribution of PCBs at the site, to evaluate the potential off-site movement of the PCBs, to assess the risks PCBs at the site might pose to human

health and the environment, and to identify remedial measures that might be needed. This work has been completed with the conclusion that the residual PCBs at the TRW site in Minerva, Ohio, present a negligible hazard to public health and the environment. TRW nonetheless requested preparation of a remedial action plan for the site. The results of the Clement study and the remedial action plan are presented in this report.

At the start of the study, more than 500 samples of soil and sediment were collected and analyzed for PCBs. The sediments and surface waters of South Pond, West Lake, and the West Lake drainage stream were also sampled and analyzed for PCBs. Additionally, 22 groundwater wells were installed and sampled seasonally for PCBs and groundwater level. Next a series of environmental transport models (including computerized predictions of groundwater movement) were utilized to identify areas of concern and to trace probable PCB migration routes to determine how humans and the environment might be exposed to the residual concentrations of PCBs at the TRW site. These exposure models were constructed using conservative assumptions to emphasize safety.

Significant residual concentrations of PCBs in soils and sediments were found in five areas of the site. In the swale (a surface drainage channel that runs south along the drum storage area on the eastern side of the plant and then westward to empty into the drainage stream discharging from West Pond), PCB concentrations ranging from less than 1 parts per million

(ppm) to 1,600 ppm were detected. Most of the residuals were found in the top 2 feet of the soil and typically ranged from 200 to 300 ppm. In the corner section of the swale, closest to where leaks and spills of PCBs are thought to have occurred, residual PCBs were found at greater depths. Concentrations exceeding 50 ppm were found at depths of 10 feet in 15% of 13 samples taken at these depths. However, the average concentration of PCBs at these depths in this segment of the swale was 16 ppm. In summary, significant PCB residuals in the swale are confined to the top 2 feet of soil and to irregular patches at deeper levels in a small segment of the swale.

In the South Pond (an evaporation pond for wax wastes generated by the plant), PCB concentrations ranging from less than 1 ppm to 2,000 ppm were found in upper layers of sediments. The average concentration in these sediments is 300 ppm. Pond sediments range in depth from 0.5 to 1.5 feet, and concentrations of PCBs in the sediments decrease markedly with depth. Thus, PCB residuals in the South Pond are confined principally to the upper layers of sediment.

In the wax ditch (a narrow channel that conveys waxy materials from the plant to South Pond), PCB concentrations ranging from 2,000 to 5,000 ppm and averaging 4,000 ppm were found in the waxy residues deposited on the bottom of the ditch. These high concentrations are explained by the high organic content of the waxy residues, which have a large adsorptive

capacity for PCBs. Low concentrations of PCBs were evident in the soils underlying the waxy residues.

In the rubble pile, (which contains asphalt, broken concrete and other debris, largely from prior construction activities) PCB concentrations ranged from less than 1 to 1,000 ppm but averaged 100 ppm. The occurrence of these residuals is patchy, and in many cases they appear to be residues in waxy materials or residues adhering to debris in the pile. These residues are largely confined to the top 2 feet of debris and soils in the pile.

Finally, in a swath of surface soils on the Fry property (owned by TRW and immediately downgradient from the South Pond), PCB concentrations averaging less than 10 ppm were found, although 2 of 51 samples exhibited concentrations greater than 10,000 ppm. These residuals are confined to the top 6 inches of soil.

Elsewhere on the site, with the exception of a half dozen samples, PCB concentrations were found to be less than 2 ppm.

The pattern of PCB concentrations found in the soil and sediment on the site clearly suggests the following:

- o The source of PCB residuals was leakage or spillage from outdoor drums of spent PCB oils that were stored in the past on the concrete pad along the east side of the plant, particularly near the southeast corner of the plant.
- o Over time, surface runoff carried by the swale has distributed PCB residues from these leaks and spills along the length of the swale. Infiltration has caused penetration of PCB residuals into the top 2 feet of the swale soils.

- o Precipitation, infiltration, and perhaps past construction activities associated with extensions of the plant have caused the penetration of PCB residuals to depths of 10 feet in the section of the swale near the southeast corner of the plant. Infiltration has caused some lesser penetration of residuals to depths of 4-5 feet in other small segments of the swale. Otherwise, PCB residuals are confined to the upper 2 feet of the soils in the swale.
- o The viscous waxy material in the wax ditch (discarded from the plant) has adsorbed PCBs from discharges into the ditch of wastes containing PCBs from spills and leaks. These materials then migrated into South Pond contributing to residual concentrations of PCBs in the pond sediments.
- o Construction activities associated with expansion of the plant, including the drum storage area, have produced debris containing PCB residuals that was placed in the rubble pile. This is likely the cause of PCB residuals found in this area.
- o Past overflows or other discharges from the South Pond have created surface soil PCB residues on a limited area of the Fry property.

These events seem to explain the patterns of PCB residues found in soils and sediments on the site and also explain the absence of PCBs in other areas of the site.

Of 22 monitoring wells installed at the site, only one well has consistently exhibited PCB concentrations exceeding the limit of detection by a significant margin. PCB concentrations in this well have ranged from 100 parts per trillion (ppt) to 1 part per billion (ppb) and average 300 ppt. However, contaminated sediments have also been detected in the well; this may contribute to the apparent water concentrations observed. Filtered water samples from this well exhibit less than half the concentrations found in unfiltered samples. This indicates

that the higher well concentrations observed may be due to sediment migration from hot spots in the local surface soil. Studies to determine the nature of PCB concentrations in the well are continuing.

PCB concentrations of 7 ppb were found in the waters of South Pond. These levels are probably caused by dissolution of PCBs from the sediments in the bottom of the pond. PCB concentrations in West Lake and its drainage stream were found to be less than 1 ppb. In summary, surface waters, except those in South Pond, do not appear to be contaminated by PCBs.

Given these findings, the question became what remediation, if any, should be undertaken to reduce the residual levels of PCBs on the site. A review of the literature and other clean-ups of PCB spills produced no clear consensus as to safe levels of PCBs in soils. Clement concluded that it was possible and appropriate to perform a risk assessment to ascertain whether the PCB residuals observed would migrate off-site and produce unacceptable exposures and risks to human health and the environment. Clement further concluded that, if unacceptable risks were found, follow-on risk assessments could be used to judge various remedial programs.

In performing these risk assessments, various pollutant transport and dispersion models were employed to predict both the worst-case and probable-case migration of PCBs from the five source areas, via air, groundwater, and surface water pathways to current or potential points of exposure.

For the air pathway, volatilization and dispersion of PCBs from the soils and the waters of South Pond was modeled to determine exposures to on-site workers and the off-site public.

For the groundwater pathway, solubilization of PCBs by precipitation infiltration and by groundwater, followed by transport of such PCBs via groundwater flow was modeled to determine exposure through drinking water consumption of persons served by the Minerva public drinking water well, persons who might use Sandy Creek as a drinking water source and persons who might, in the future, install and use private drinking water wells placed in the path of potential PCB migration from the site.

For the surface water pathway, potential transport of PCBs and PCB-laden sediments from the site to Sandy Creek was assessed. To translate predicted exposure levels into a risk evaluation, the scientific literature on the health effects of PCBs was thoroughly studied to ascertain threshold levels for noncarcinogenic health effects and dose-related risk levels for carcinogenic effects. These findings were combined with predicted exposure assessments to provide an assessment of potential risks.

In both the modeling of migration and dispersion of PCBs and the follow-on assessment of risks, various assumptions had to be made about the quantity of precipitation, the permeability of soils and sediments, the rate of volatilization



and other factors. In all cases (for both the worst-case and probable-case assessments) very conservative assumptions were used in order to overstate the predicted risks and thereby err on the safe side.

The results of the worst-case and probable-case risk assessments revealed that the current situation produced very small risks and acceptable margins-of-safety against current or future health and environmental effects. Consequently, the risks presented are not considered sufficient to require any remedial action. Notwithstanding, the potential risks to on-site workers and potential users of future private wells, although very small, were found to be slightly higher than other risks. TRW, desiring to provide the highest levels of safety to the public and its workers, requested development of a remedial action plan to reduce these risks even further--to insignificant levels. In response, Clement Associates designed the remediation program which is presented in this report.

The program has three principal elements. First, the removal of all surface PCB residuals is recommended to virtually eliminate potential exposure via air and surface water pathways (i.e., through volatilization and erosion). It is recommended that this be accomplished by:

1. Under an NPDES permit, draining, treating, and discharging all the water in South Pond.
2. Removing and disposing all of the sediments in South Pond.
3. Removing and disposing the top 18-24 inches of soil from the swale.

4. Removing and disposing all of the wax residues and the top 1 foot of underlying soils from the wax ditch.
5. Removing and disposing the debris and the top 6-12 inches of soil from the rubble pile.
6. Removing and disposing the top 6 inches of soil from the area of the Fry property exhibiting significant PCB residues.
7. Replacing removed material with clean soil in South Pond, the wax ditch, and the Fry property and replacing removed material with compacted clay (hence a clay cap) in the swale and the rubble pile.
8. Grading and planting the areas with grass to promote drainage and prevent erosion.

Second, it is recommended that the soils and sediments removed be disposed of in a secure on-site cell that is designed, constructed, and operated in compliance with the Annex II requirements of 40CFR761.71. In this way, ultimate deposition of PCB residuals will be as safe, if not safer, than off-site landfill disposal and will ensure the safe, permanent interment of these materials.

The third element of the plan is included in the first element. It involves removing the major amounts of PCB residuals from the soil column in the areas where PCBs are found at depths below 2 feet, and capping these areas with compacted clay. The purpose of this element is to minimize precipitation infiltration and thereby significantly reduce potential exposures through the groundwater pathway.

Additionally, the plan recommends continued monitoring of groundwater, surface water, and sediments during and after the remedial actions.

To assess the accomplishments of the remedial plan, a worst-case risk assessment was made for the after-remediation situation. This assessment shows insignificant exposures and risks through all three pathways, from all five sources. As such, the recommended remedial plan produces an exceptionally high margin-of-safety against any current or future adverse health or environmental effects.

## I. INTRODUCTION

In early August 1981, TRW personnel discovered the presence of an area of soil contaminated with polychlorinated biphenyls (PCBs) at the southeast corner of their plant in Minerva, Ohio. Plant records suggested that spent oil used in diffusion pumps that contained PCBs (principally Aroclor 1254) had been stored in this area, so that further investigation was warranted. Since use of such oils was discontinued in 1973, there is no longer a source of PCB contamination at the site. Thus, attention was focused on residual levels of PCBs that might exist in the soil and sediment on the TRW property. TRW personnel notified appropriate officials within the U.S. Environmental Protection Agency and the Environmental Protection Agency of Ohio. To date, both agencies have been supportive of TRW's remedial action efforts and have stated publicly that residual PCB concentrations at the site do not appear to pose an imminent hazard to the surrounding populace. Limited soil and water sampling was conducted by EPA and Ohio EPA, as well as TRW. Results suggested that soil contamination had occurred throughout a swale used to control site runoff and that other areas on the property also might contain residual concentrations of PCBs.

In October 1981, TRW retained Clement Associates to assess hazards associated with the PCB contamination at the site and to assist TRW in developing remedial measures for mitigating

such contamination. Toward this end, a multifaceted study was initiated to:

- o Determine the extent and distribution of PCBs at the site
- o Evaluate the movement of PCBs at the site
- o Assess any health and environmental effects associated with PCB exposure
- o Assess any risks to human beings and the environment from the residual concentrations of PCBs found at the site
- o Identify technologies used to clean up PCB contamination
- o Evaluate the applicability of such technologies to the specific situation at the Minerva site
- o Evaluate and select appropriate technological options to be incorporated in a remedial action plan for cleaning up the site, if such action was deemed advisable based on the risk assessment.

The Clement study proceeded in phases. First, a program of sampling and analysis was conducted to characterize residual PCB concentrations at the site. The history and characteristics of the Minerva site were evaluated to facilitate design of the sampling program for determining in detail the level and distribution of PCBs on the property. As sampling progressed, a preliminary transport model was developed to trace probable PCB migration routes and to identify areas of concern. Based on this model, a groundwater monitoring program was instituted, and soil, sediment, and surface water were analyzed. In an iterative process, sampling results were used to refine the model, and the model provided a basis for targeting further sampling. At a later stage, the model was also used to predict

future movement of PCBs so that attendant risks could be evaluated.

In the second phase, a risk assessment was performed to evaluate the potential health and environmental effects resulting from the residual levels of PCBs found at the site. This assessment was intended as a guide for determining the need for remedial measures to protect the health of individuals in the vicinity of the site and to maintain the integrity of the local environment. Thus, the potential for the migration of PCBs from the site, the potential for exposure due to the migration of PCBs and the potential health and environmental consequences due to possible exposure were each evaluated as part of this assessment.

Remedial measures were considered in the third phase of this study. Various proven and experimental remedial alternatives were evaluated for applicability and effectiveness. A remedial action plan was developed based on results of this evaluation.

Results and conclusions from each phase of this study are presented in the following chapters of this report.

## II. CHARACTERIZATION OF RESIDUAL PCB CONCENTRATIONS

Soils, sediments, surface water, and groundwater at the site were all sampled as part of an extensive program to characterize the distribution of residual PCBs at the site. Augmenting the sampling program, environmental models were employed to evaluate runoff, evaporation, infiltration, and dispersion from those regions on the property where residual levels of PCBs were detected. Over 500 soil samples were collected and analyzed for PCBs. The distribution of PCBs in sediments and surface water was characterized from over 100 samples. Twenty-two groundwater monitoring wells were installed, and samples from the wells are collected and analyzed monthly. The potential for groundwater contamination was also modeled with the aid of a two-dimensional, finite-difference computer program. Factors and site characteristics affecting the migration of PCBs are described in the following site description. Results of the sampling and analysis program and environmental modeling are summarized in a profile of residual PCB concentrations.

### A. Site Description

The TRW Metals Division site at Minerva, Ohio, depicted in Figure II.1, is situated on a relatively flat 54-acre tract of land. There are two retention lagoons, one of which receives treated plant wastewater, a large pond (South Pond) that has received a variety of plant effluent in the past, and a drainage swale that protects the plant from flooding and passes a drum

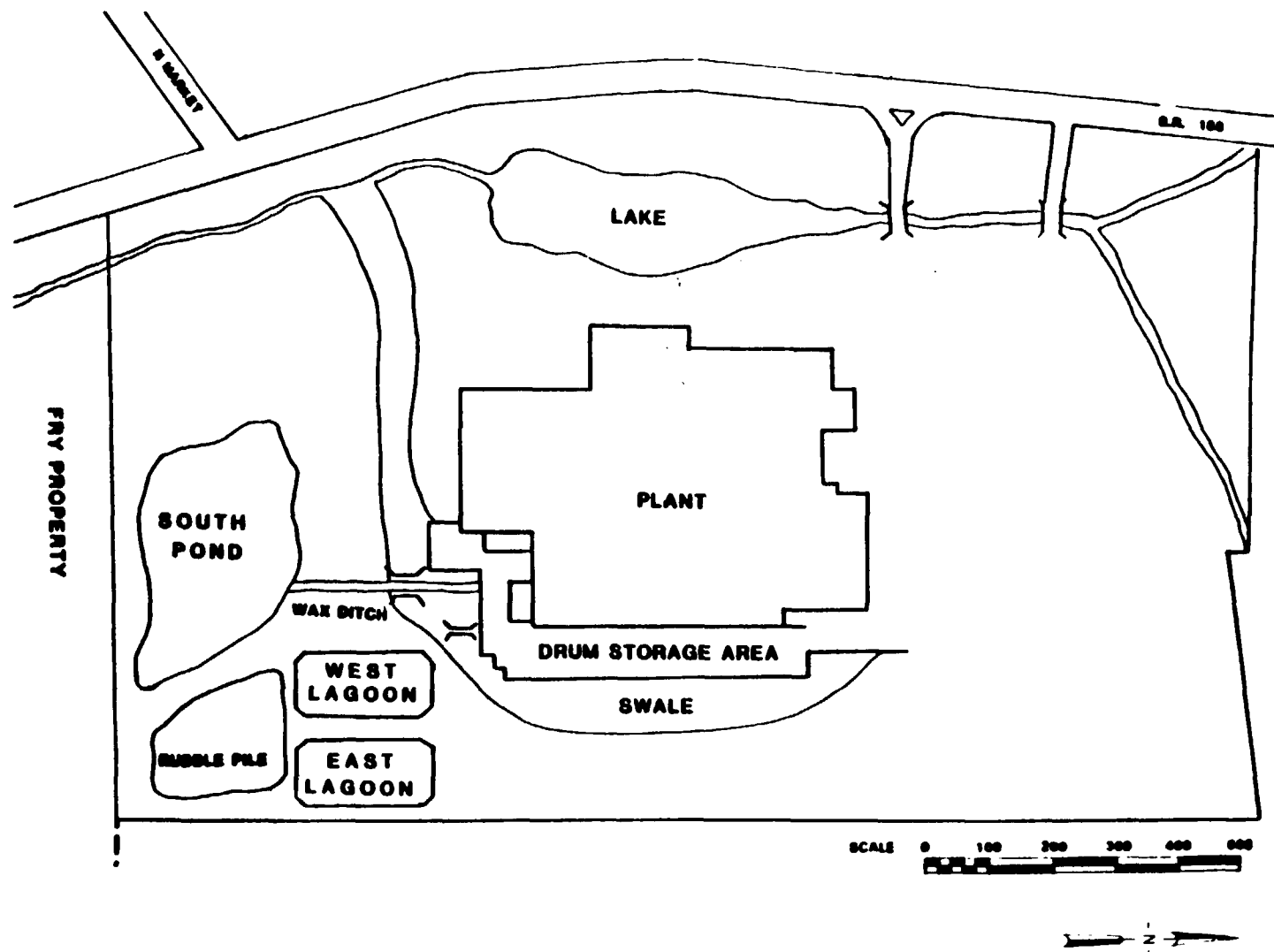


FIGURE II.1

LAYOUT OF THE MINERVA SITE



storage area. A natural stream running along the western edge of the property was dammed to create an ornamental lake (West Lake). The drainage swale accumulates rainwater runoff and discharges to the stream emanating from West Lake. Discarded casting wax and other plant effluent were transported to South Pond via a 300-foot ditch (wax ditch). South Pond loses water principally via evaporation; the pond does not discharge to surface water and core sampling reveals the presence of a clay lining that restricts percolation to groundwater.

The TRW property is bounded on the north and east by level farmland. On the west, the land slopes upward from the property boundary on Iowa Avenue. Southward, following an abrupt drop in elevation of 4-5 feet in the vicinity of South Pond, the land slopes gently and irregularly down through a wooded area (the Fry property) to Sandy Creek, which is 700 feet from the pond. Thus, because of the local topography, surface drainage on the unmodified portions of the property is generally to Sandy Creek. TRW purchased the Fry Property in 1982 for the purpose of locating monitoring wells. The town of Minerva lies beyond Sandy Creek, south and west of the plant (Figure II.2).

Features of the upper strata and the upper aquifer in the vicinity of TRW are relatively uncomplicated. The facility is situated on glacial till composed of a well-drained soil classified as Chili silt loam (USDA 1980). The upper 2-4 feet of these soils are loamy, but they become increasingly sandy and gravelly with depth. The glacial till is 90 feet thick

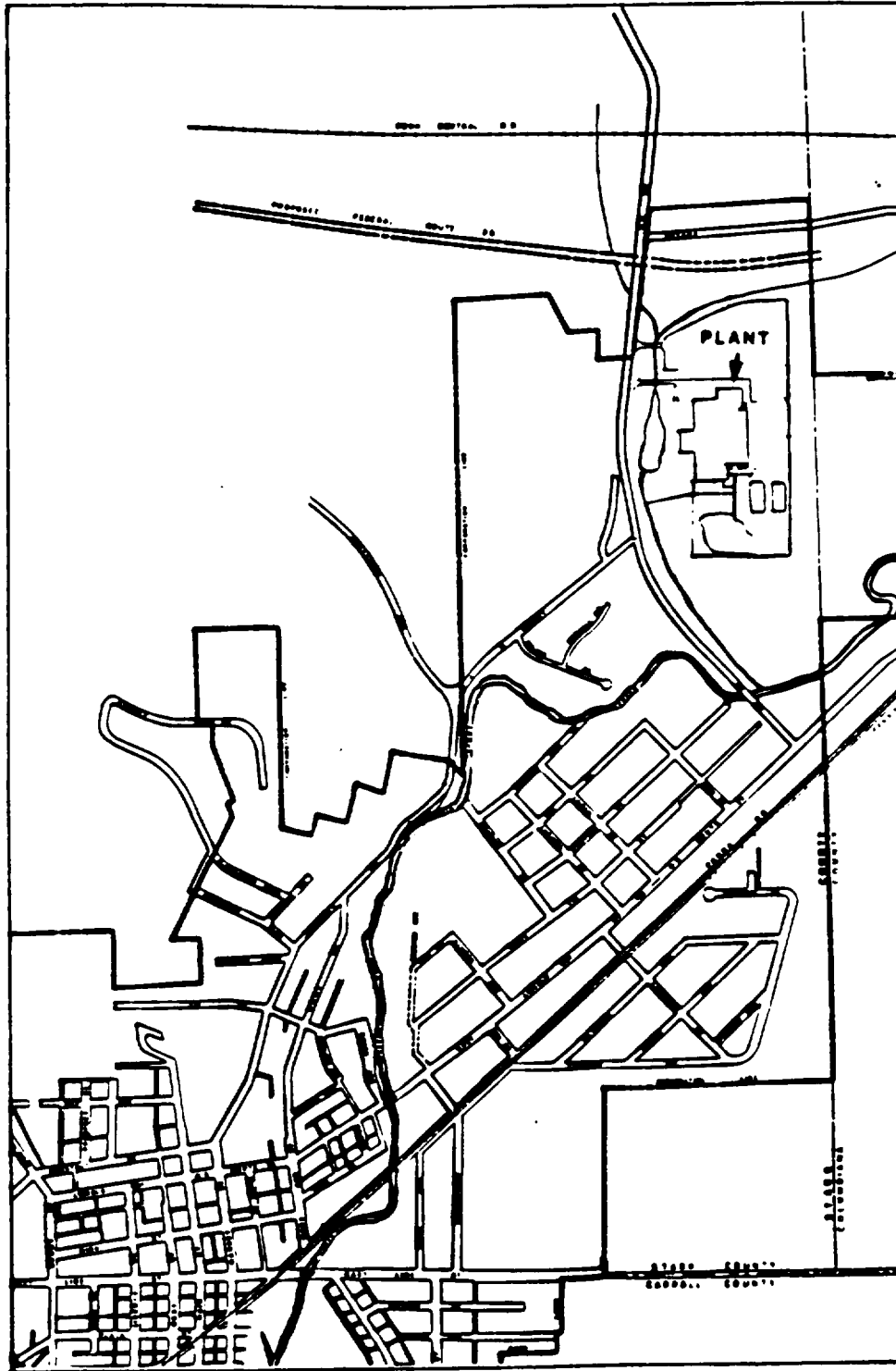


FIGURE II.2

VILLAGE OF  
MINERVA, OHIO

LOCATION OF THE PLANT

FEBRUARY, 1982



in this area and is underlain by bedrock consisting of shale, slate, clay, and limestone.<sup>1</sup> The groundwater table varies seasonally in this region by several feet. Based on a seasonally adjusted average, groundwater is encountered on the site at depths ranging between 5 and 12 feet. Groundwater flows to the south. A schematic of the geologic strata and the aquifer underlying the TRW property is presented in Figure II.3.

#### B. Profile of Residual PCB Concentrations

A master sampling map was developed to facilitate communication between Clement Associates and the sampling team from Alert Laboratories,<sup>2</sup> and to allow detailed tracking of the progress of data collection. The base of the map is a scale representation of topological features and artificial structures on the site. Sampling results are depicted on a series of overlays. A reduction of the base map and the master key index, which is a compilation of all sampling results, are presented in Appendix A.

Results of the sampling and analysis program reveal that most residual PCBs at this site are distributed among four distinct topographical features: the swale, South Pond, the wax ditch, and a rubble pile. The only other area where potentially significant levels of PCBs were identified is a narrow

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<sup>1</sup>Based on a survey of local well logs

<sup>2</sup>Alert Laboratories (Box 208, Canton, OH 44701) is a subcontractor to both Clement and TRW and has performed most of the sampling and analysis at the Minerva site.

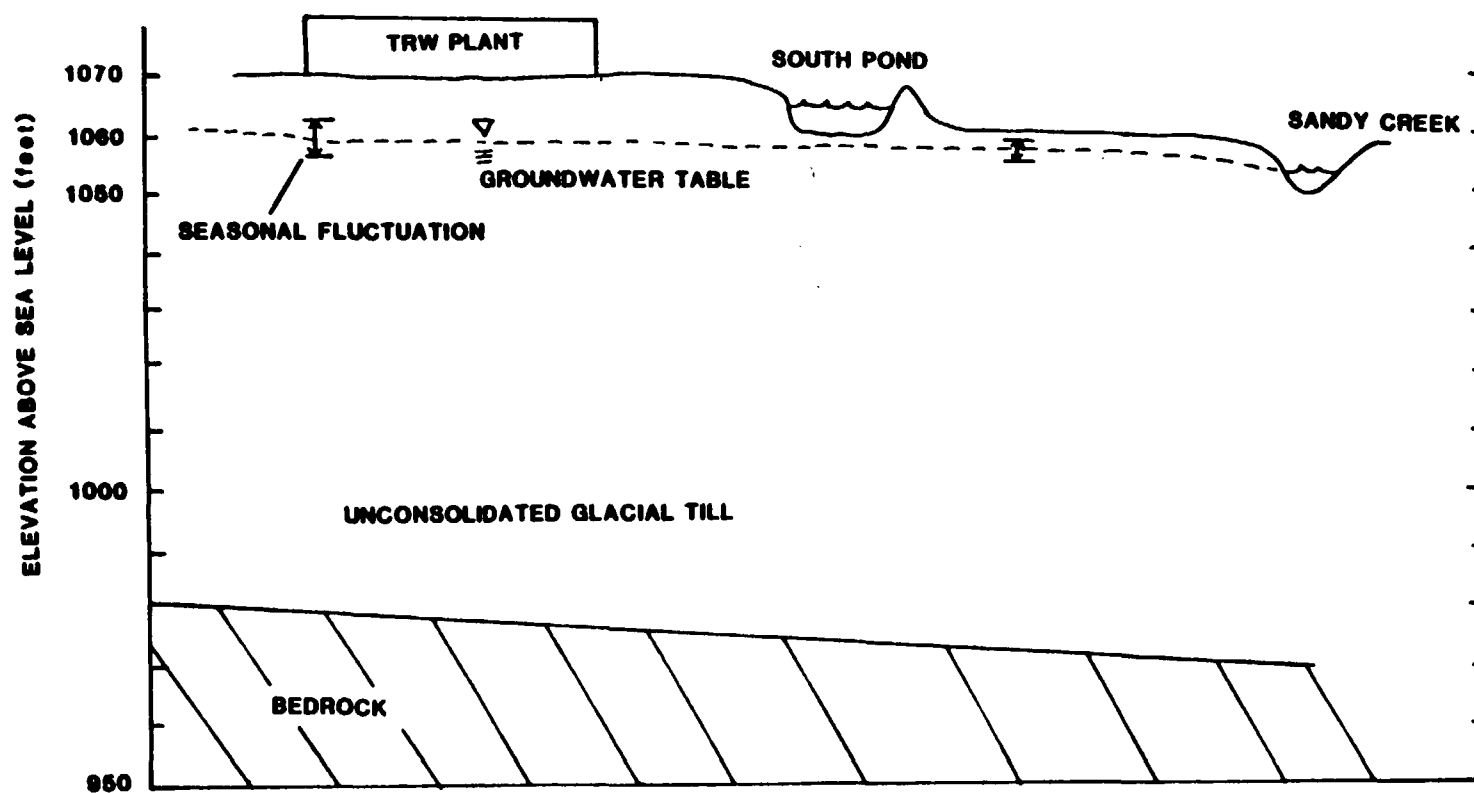


FIGURE II.3

GEOLOGIC AND HYDROLOGIC FEATURES OF THE UPPER STRATA  
UNDERLYING THE TRW MINERVA FACILITY

swath of surface soil on the Fry property (owned by TRW) in the vicinity of South Pond.

PCBs are unevenly distributed in the swale. Concentrations as high as 1,600 ppm have been detected near the former drum storage area at the southeast corner of the plant, although typical concentrations for swale soils lie between 200 and 300 ppm. PCB concentrations decrease sharply with increasing depth under most of the swale. Generally contamination above 50 ppm is confined to the first 1-2 feet of soil. In the corner area of the swale, however, concentrations exceeding 50 ppm have been detected at depths of 10 feet in 15% of the 13 deep samples collected. The average concentration of samples from these depths is 16 ppm.

PCBs are present in both the water and sediment of South Pond. The concentration of PCBs in South Pond water averages 7 ppb<sup>1</sup> and varies slightly with depth and location. The distribution of PCBs in South Pond sediment is highly irregular with localized hot spots as high as 2,000 ppm. Typical values for the top layers of sediment are close to 300 ppm. Concentrations decrease with increasing sediment depth.

A waxy organic residue covers most of the bottom of the wax ditch. PCB levels average 4,000 ppm in this material.

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<sup>1</sup>The solubility of PCBs decreases with increasing chlorine content. Based on gas chromatographic analysis, the spilled material is believed to consist largely of Aroclor 1254, which is a mixture of tetra-, penta-, and hexachlorinated isomers. (A fraction of the spill may have consisted of Aroclor 1242 containing more highly soluble trichlorobiphenyls.) The solubility of such isomers varies from 10 to 200 ppb with 50 ppb representing a weighted average (Griffin and Chian 1980).

Except for isolated samples from other areas containing residual levels of PCBs, the waxy residue in this ditch represents the most highly contaminated material on the site.

The rubble pile, a mound of material east of South Pond, contains asphalt, broken concrete, waxy residues, and other debris. Some of the material is contaminated with PCBs. PCB concentrations in this area are highly irregular; 3 of the 89 samples collected in this area contained up to 1,000 ppm. Average concentrations throughout the breadth and depth of the pile, however, are closer to 100 ppm.

Residual PCBs on the Fry property exist only at the surface. The distribution of PCBs consists of a disperse series of localized hot spots lying in a poorly defined band running southwest from the edge of South Pond. Peak concentrations at hot spots decrease with increasing distance from South Pond. Although 2 of 51 samples from this area exhibit concentrations exceeding 10,000 ppm, the average concentration of other samples from the Fry property is less than 10 ppm.

Residual PCB concentrations over remaining areas of the site are minimal. Regions adjacent to areas of higher residual concentrations exhibit levels of a few ppm. The level in one sample from West Lake sediment exceeded 50 ppm, although typical values for sediment in West Lake are less than 10 ppm. Other than one or two isolated sampling points, PCB concentrations exhibited over the remaining site were below the level of detection.

An overview of residual PCB concentrations at the site is shown in Table II.1. Figures II.4, II.5, and II.6 present residual PCB concentrations in soils and sediments at depths of up to 6 inches, 6 inches to 2 feet, and more than 2 feet, respectively. The shaded areas in these figures distinguish between concentrations of less than 50 ppm, 50-200 ppm, and more than 200 ppm. These figures readily illustrate that PCB concentrations decrease with depth and that most of the residual PCBs on the site are contained within the first 1-2 feet of soil.

Concern for possible groundwater contamination led to placement of monitoring wells and analysis of local groundwater for the presence of PCBs. The site was surrounded initially with 18 wells to test for contamination and determine the direction of groundwater flow. As the monitoring program progressed, however, it became evident that additional sampling points would be required to determine if groundwater contamination was, in fact, present. As a result, four additional wells were placed further downgradient in November 1982. Groundwater levels measured in all wells are presented in Table II.2. Typical groundwater level contours are depicted in Figure II.7. Results of PCB sampling and analysis in groundwater are presented in Table II.3.

Of the 22 wells installed on site, only well 10 has consistently exhibited PCB concentrations exceeding the limit of detection by a significant margin (Table II.3). Well 10, which is located on the Fry property 150 feet from South Pond, is

TABLE II.1

## OVERVIEW OF RESIDUAL PCB CONCENTRATIONS AT THE TRW SITE

Region	Surface Area (ft <sup>2</sup> )	Volume (ft <sup>3</sup> )	Typical Concentrations (ppm)	Highest Level Found (ppm)	
South Pond sediment	7.0x10 <sup>4</sup>	7.5x10 <sup>4</sup>	300	2,000	Concentrations decrease with depth
Swale	7.5x10 <sup>4</sup>	2.2x10 <sup>5</sup> <sup>a</sup>	250	1,600	Concentrations decrease with depth (but there is contamination at 10 feet in some spots)
Wax ditch	3.0x10 <sup>3</sup>	7.5x10 <sup>3</sup>	4,000	5,000	Contamination concentrated in organic residue
Rubble pile	(2.3x10 <sup>4</sup> ) <sup>b</sup>	(9.2x10 <sup>4</sup> ) <sup>b</sup>	100	1,000	Area and composition are not well defined
Fry property	(1.2x10 <sup>5</sup> ) <sup>c</sup>	(1.3x10 <sup>4</sup> ) <sup>c</sup>	<10 <sup>c</sup>	33,000	Area and composition are not well defined

<sup>a</sup>Depth of soil containing residual PCBs depends heavily on concentrations considered. This volume is for soil suspected to contain to at least 50 ppm.

<sup>b</sup>Because the rubble pile is an amorphous conglomeration of material, it is poorly defined; the area and volume are thus estimates.

<sup>c</sup>Residual PCBs on the Fry property are irregularly distributed. The volume and surface area represent estimates of the region where PCBs occur in concentrations greater than 50 ppm. In this case, typical and average concentrations do not correlate.



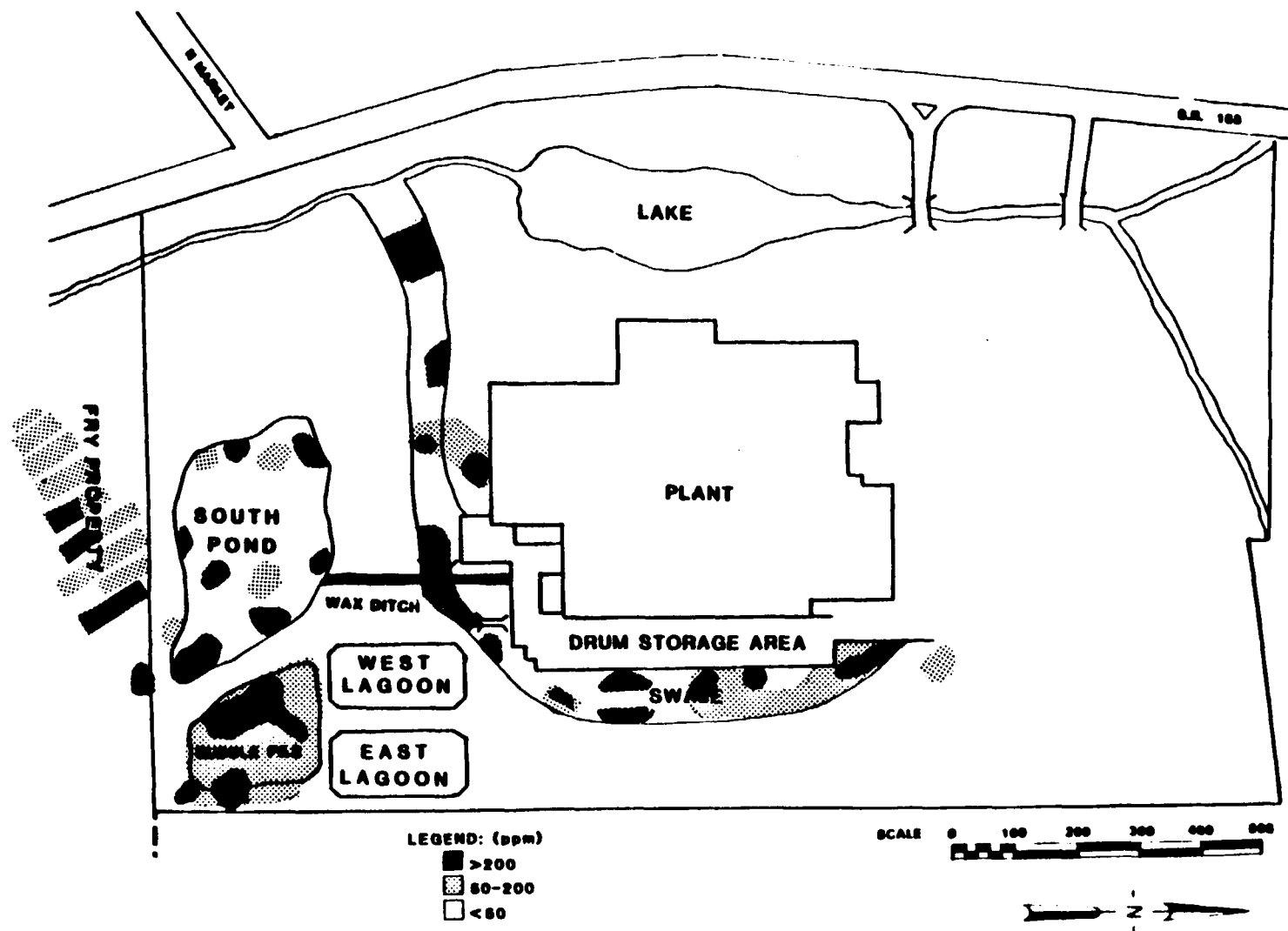
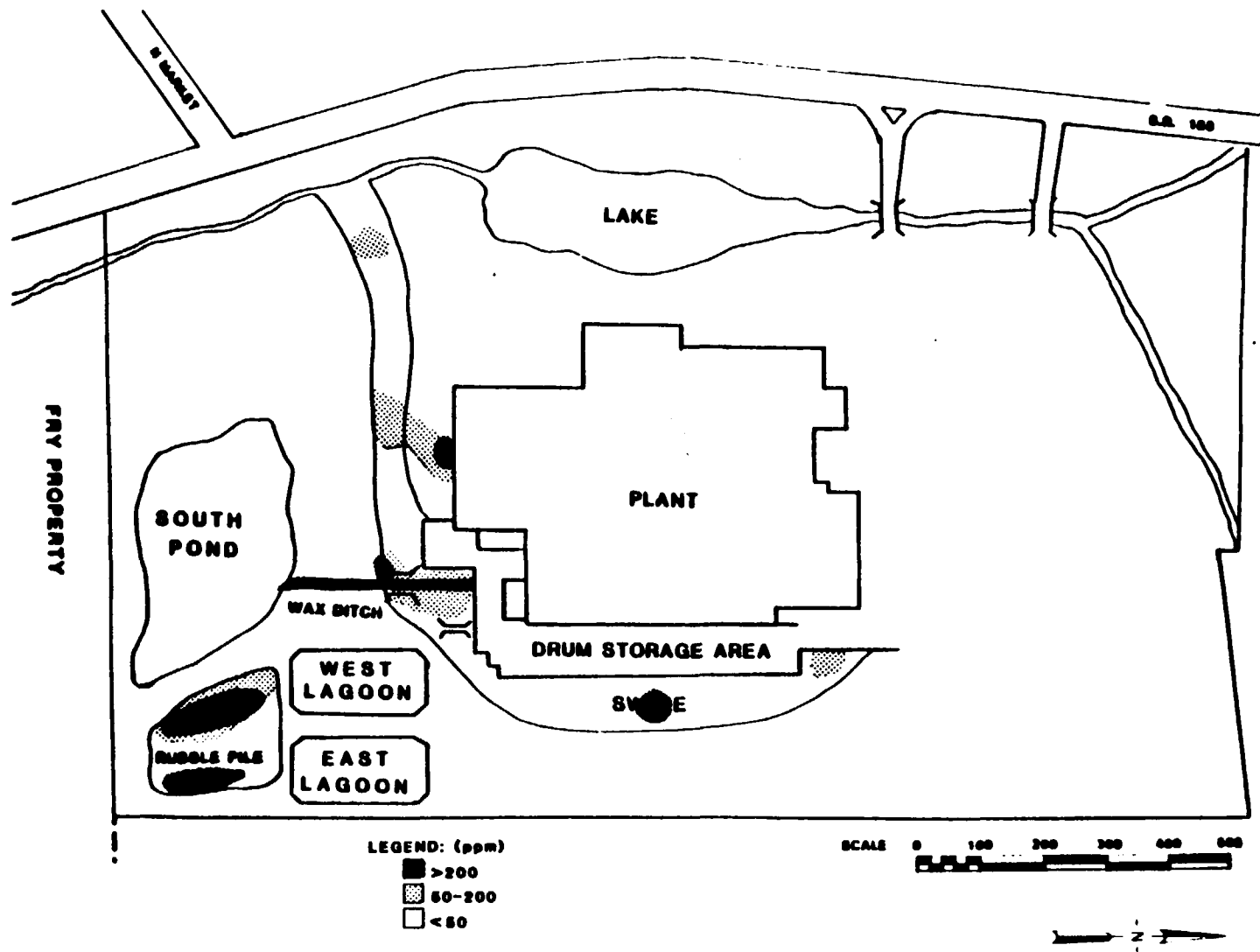


FIGURE II.4  
PROFILE OF PCB RESIDUALS AT THE TRW MINERVA SITE  
DEPTH 0-6"



**FIGURE II.5**  
**PROFILE OF PCB RESIDUALS AT THE TRW MINERVA SITE**  
**DEPTH 6"-2'**

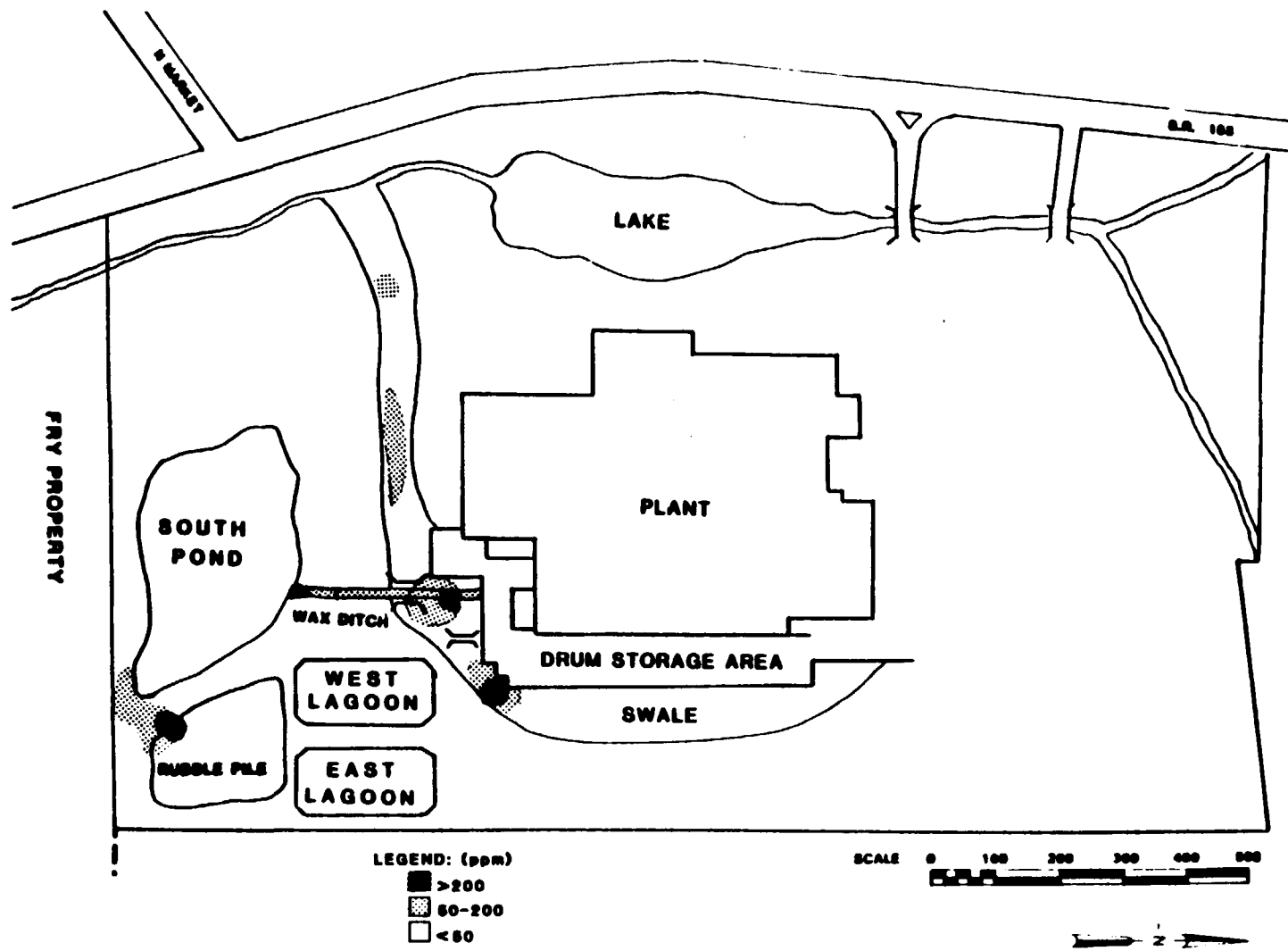


FIGURE II.6  
PROFILE OF PCB RESIDUALS AT THE TRW MINERVA SITE  
DEPTH BELOW 2'

TABLE II.2

NORMALIZED WATER LEVELS OF WELLS AT SITE  
(depths in feet below top of well No. 9)

Well	Sampling Dates											
	4/81	8/13/82	9/1/82	10/8/82	10/28/82	11/15/82	11/23/82	12/14/82	1/4/83	2/18/83	3/30/83	4/27/83
1	1.21	4.45		5.01	5.12	4.90	4.73	4.89	3.61	3.24	1.77	1.20
2	1.35	4.68		5.21	5.32	5.04	4.61	5.03	3.84	3.64	2.72	1.97
3	1.59	4.43		4.92	5.15	4.87	4.65	4.84	3.80	3.66	2.95	2.20
4	1.35	3.52		4.11	4.39	3.80	3.41	3.78	3.01	2.76	2.26	1.78
5	2.84	5.62		6.18	6.30	6.13	5.98	6.07	4.71	4.46	3.24	1.80
6	10.03	5.86		6.34	6.51	6.32	6.06	6.29	4.93	5.11	3.90	3.13
7		5.86		6.36	6.52	6.30	6.10	6.28	5.05	4.81	3.89	3.07
8	3.75	5.91		6.29	6.41	6.26	5.99	6.23	5.20	5.08	4.39	3.97
9	4.59	6.58		6.85	7.08	6.89	6.56	6.91	5.96	6.02	5.24	4.92
10	4.50	7.28		7.59	7.69	7.51	7.29	7.17	6.56	6.45	5.75	5.18
11	5.57	7.15		7.48	7.57	7.41	7.18	7.17	6.46	6.32	5.58	4.98
12	5.61	7.13		7.51	7.56	7.36	7.16	7.13	6.36	6.28	5.27	4.84
13	4.35	6.77		7.17	7.27	7.08	6.84	7.05	6.04	5.84	5.02	4.50
13a	2.35	6.53		6.86	6.86	6.68	6.62	6.66	5.53	5.21	4.58	3.95
14	4.18	6.24		6.61	6.71	6.53	6.33	6.52	5.45	5.33	4.51	3.83
15		5.18		5.63	5.68	5.51	5.32	5.49	4.25	4.00	3.32	2.57
16		5.13		5.60	5.71	5.52	5.34	5.50	4.26	4.20	3.38	2.41
17		4.84		5.31	5.42	5.22	5.05	5.21	4.07	3.88	3.08	2.21
18							7.24	7.27	6.52	6.37	5.69	5.21
19							7.58	7.62	6.91	6.70	6.15	5.58
20							7.63	7.65	6.96	6.80	6.08	5.56
21							7.22	7.21	6.49	6.32	5.58	5.02
Pond		-0.22				-0.01			-0.26		+0.05	
Composite												
water level												
(average)		5.73		6.17	6.29	6.07	5.85	6.01	4.95	4.79	3.94	3.26
Rainfall												
since previous												
sampling date												
(inches)				1.22	0.34	1.37	1.60	1.87	--	1.30	2.25	2.68

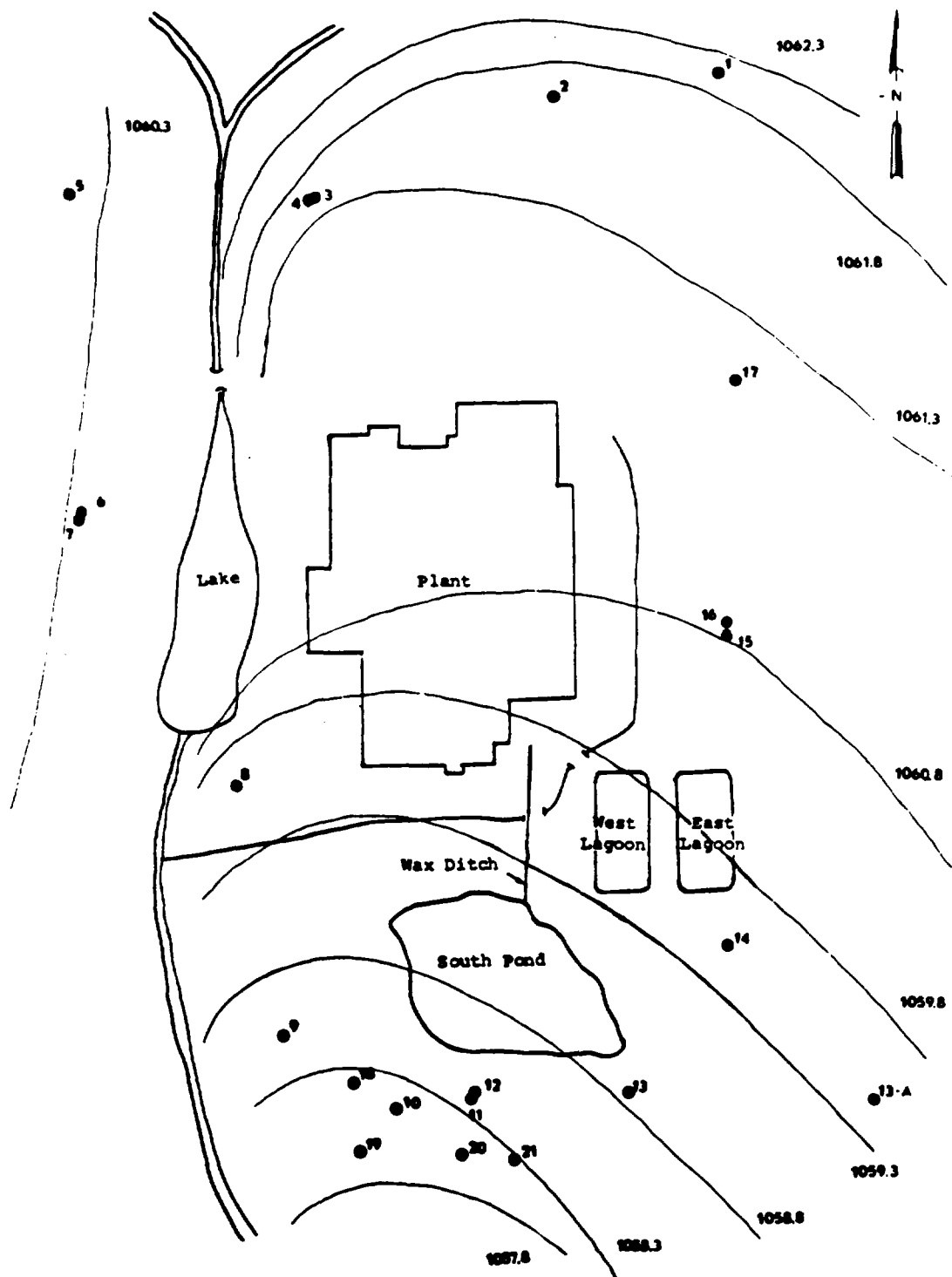


FIGURE II. 7

MONITORING WELL PLACEMENT AND GROUNDWATER CONTOURS

(1/4/83)

TABLE II.3  
PCB CONCENTRATIONS IN WELL WATER SAMPLES (ppt)

Well	Sampling Dates											
	Initial 8/11	8/11 <sup>a</sup>	9/1-9/3	10/8	10/28	11/14	11/23	12/14	1/4	2/18	3/30	4/27
1	20	20	20	20		20	20	60 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>
2	20	20	20									
3	22	28	20									
4	20	20	78		100	35						
5	20	20	20									
6	20	20	20									
7	20	20	20									
8	26	20	25									
9	20	20	20	20	100	55	20	40 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>		
10	364	462	1,020	770	560	240	360	390, 760 <sup>a</sup>	370, 150 <sup>c</sup>	340 <sup>a</sup> , 340 <sup>a, d</sup>	180 <sup>a, e</sup> , 340 <sup>a</sup>	410 <sup>a</sup> , 320 <sup>a, c</sup>
11	20	20	65	40	120	60	55	50 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>
12	72	105	165	180	180	155	25	80 <sup>a</sup>	80 <sup>a</sup>	50 <sup>a</sup>	65 <sup>a</sup>	509
13	97	20	20	30	140	20	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>		
13a	20	20	20				20					
14	95	25	20									
15	20		20									
16	414	76	20	20								
17	20	23	20									
18							20, 20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	30 <sup>a</sup>		
19							60, 50 <sup>a</sup>	40 <sup>a</sup>	20 <sup>a</sup>	45 <sup>a</sup>	30 <sup>a</sup>	70 <sup>a</sup>
20							30, 20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>
21							25, 20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>	20 <sup>a</sup>		
Pond									4500	7400 <sup>f</sup>		

<sup>a</sup>After evacuation ?

<sup>b</sup>Yellow water at top, clear in middle, yellow at bottom--no odor

<sup>c</sup>After filtering

<sup>d</sup>Duplicate

<sup>e</sup>After extended settling

<sup>f</sup>Average of four samples from different locations

downgradient of the main section of the TRW property. Although some of the observed concentrations of PCB in the well are attributable to contaminated particulates that have accumulated in the well and are partially suspended in the water, the PCB concentration in this well peaked at an apparent level of 1 ppb on September 3. Since that time, PCB levels observed in this well appear to have stabilized at 300 ppt. Analysis reveals that the sediment in the well contains 1 ppm PCB. Filtered water samples from well 10 have exhibited less than half the PCB concentration of unfiltered samples. The exact nature of the contamination in well 10 is therefore subject to question. Studies necessary to resolve this question are currently in progress. In either case, however, well water concentrations exhibited to date are below the EPA-defined limit of detection of 1 ppb for water and, in most cases, below the EPA-defined limit of sensitivity of 100 ppt.

### III. RISK ASSESSMENT

Once the distribution of residual PCBs at the TRW site was characterized, risks associated with the potential migration of PCBs were evaluated. Initially risks were evaluated assuming that only natural forces would determine the transport of PCBs subject to the conditions at the site that currently exist. To evaluate the efficacy of remedial measures for the final phase of this study, potential risks were reevaluated based on site conditions that would prevail when remediation was completed. All potential routes of migration were considered, although it was recognized that exposure via the groundwater route would not occur for 100 years if at all. Finally, in the absence of empirical data, conservative starting assumptions were used so that the result of the analysis is likely to be far more severe than actual consequences. In other words, a "worst-case" evaluation was performed.

The risk assessment was performed in three steps. First, potential PCB migration and attendant exposure were evaluated in an exposure assessment. Then potential health effects of PCBs were evaluated in a toxicology assessment. Finally, risks were evaluated by considering the results of the exposure and toxicology assessment. Each of these steps is summarized in the following sections.



## A. Exposure Assessment

The source of PCBs at the Minerva site was removed prior to 1974. Therefore, the potential for exposure from residual PCB concentrations as they presently exist depends on the current distribution of PCBs and the effects of transport mechanisms that cause migration and may lead to exposure now and in the future. In a sense, the profile of residual PCB concentrations presented in the previous section provides a picture at a single point in time of a situation undergoing continual development. Although the exact routes of migration leading to the current distribution are not known, we do know that contamination has ceased so we are dealing solely with future dispersion of the residual material already present. Assuming the situation is not altered intentionally (by the introduction of remedial measures), future migration must be considered before the potential for exposure and attendant risks posed by residual PCBs can be fully evaluated.

Following a background discussion of transport processes that cause migration of PCBs, we present an evaluation of the potential for exposure via each of these processes. Each region where concentrations of residual PCBs have been identified is evaluated considering local geology, hydrology, meteorology, and the properties of PCBs.

### 1. Transport Processes

The migration rate of any chemical through the environment depends upon the physicochemical properties of the migrating

substance as well as the physicochemical properties of the surrounding medium. Transport processes (depicted in Figure III.1) that affect migration of PCBs from the TRW site include percolation, volatilization, and surface water runoff. These mechanisms potentially contribute to groundwater contamination, air contamination, and surface water contamination, respectively. Parameters used to determine the magnitude of exposures via these pathways and methods used to evaluate their effects are discussed below in terms of an interpretive model. The model was used with available sampling and monitoring data to assess the cumulative impact of residual PCBs as they currently exist at the site.

a. Percolation

Percolation is the transport of PCBs in a mobile liquid phase through a solid matrix such as soil or sediment. Vertical percolation is driven principally by water from precipitation. PCBs are transported either as a dissolved solute in the water phase or, in areas where PCBs have saturated the ground, as a separate organic phase pushed ahead by percolating rainwater.<sup>1</sup> In either case, the rate of transport is limited by the average yearly quantity of percolating precipitation (of 40 inches of annual rainfall in the Minerva area, 10 inches percolate).

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<sup>1</sup>A closely related mechanism that may be important at this site, is migration of PCBs adsorbed on sediments settling through a coarser soil matrix (Dr. Yaron Sternberg, private communication. Dr. Sternberg, a hydrologist, is a professor of civil engineering at the University of Maryland and an Associate Engineer with Clement Associates.

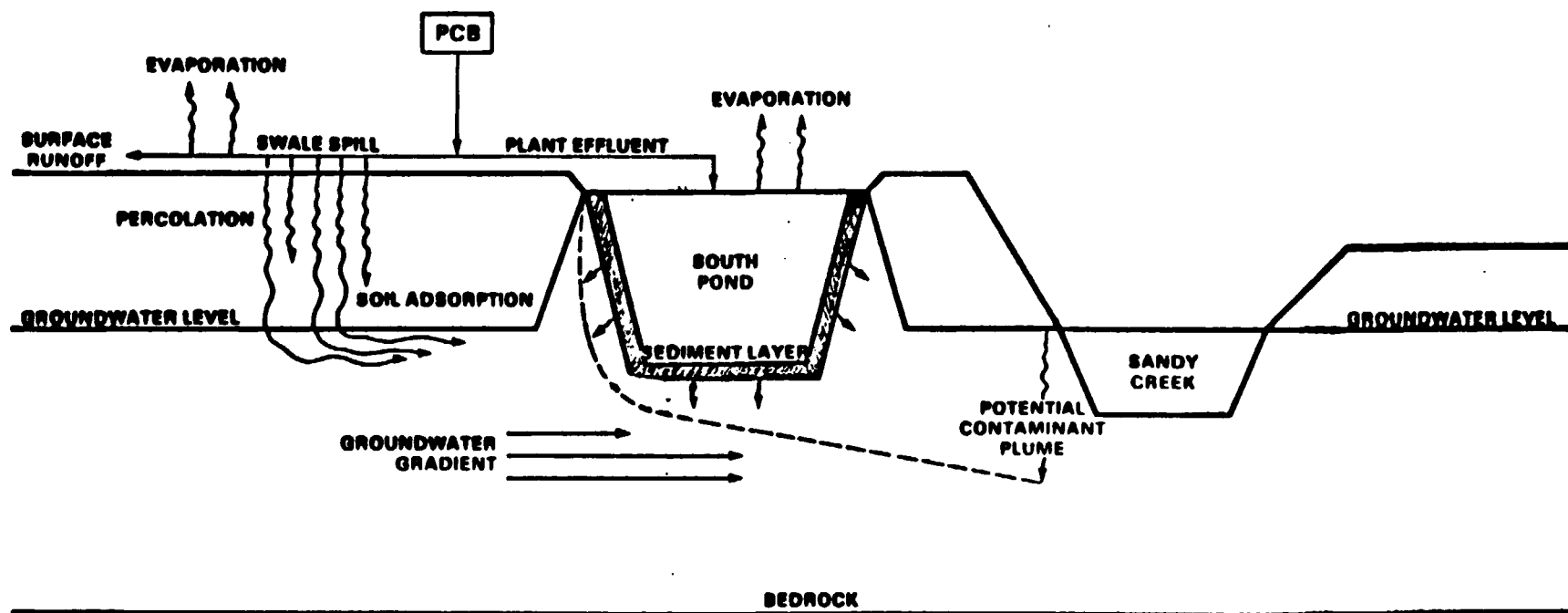


FIGURE III.1

ENVIRONMENTAL TRANSPORT OF PCBs

Once percolating PCBs reach groundwater, the material is driven horizontally by groundwater flow. At this site, dissolved PCBs are expected to be the only important contribution to groundwater transport over large distances because of the limited size of the total spill at the site.<sup>1</sup> (Supporting this hypothesis, tests for the presence of an organic phase facilitating transport of PCBs in groundwater have been negative.)

The relationship between soil permeability and percolation rate is given as:

$$Q = PIA \quad (1)$$

where  $Q$  is the percolation rate in cubic feet/day,  $P$  is the soil permeability in feet/day,  $I$  is the dimensionless pressure drop per unit distance across the soil (the driving force), and  $A$  is the cross-sectional area through which percolation occurs in square feet. (Permeability, in turn, is a function of the chemical composition and particle size distribution of the soil.) The transport of contaminants (PCBs), which are dissolved in a percolating solvent (water), will generally be slower than the rate of solvent movement because the contaminant will distribute itself between the stationary soil phase and mobile solvent phase. At equilibrium, the concentration ratio between the fraction of contaminant adsorbed in the soil and the fraction of contaminant dissolved in the solvent is known as the partition coefficient,  $K$ .  $K$  is a function of the surface

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<sup>1</sup>When a saturated PCB phase has traversed a soil volume sufficiently to permit adsorption, further transport will occur entirely via PCBs dissolved in water.

area of the soil particles, the solubility of the contaminant in the mobile solvent, and the chemical properties of both the soil and contaminant that affect their relative affinity. Typical values for permeabilities and partition coefficients of soil and other materials found at the site are presented in Table III.1. It should be noted that partition coefficients in Table III.1 are derived assuming that adsorption is completely reversible. There is evidence, however, that irreversible processes play an important role in the interactions between PCBs and soil. (See, for example, Di Toro and Horzempa 1982). Such processes are considered further in evaluating transport via percolation.

Potential movement and dispersion of PCBs in groundwater at the Minerva site was modeled with the aid of a computer. (Details are presented in Appendix B.) Briefly, the size of a contaminant plume in groundwater and the profile of concentrations within the plume are a function of the properties of the contaminant, the properties of the local soil, and the characteristics of local groundwater flow. One important conclusion drawn from groundwater modeling is that the concentration,  $C$ , at any point in a contaminant plume is a strong function of the rate of influx of contaminated water,  $Q$ , from a source and the initial concentration,  $C_0$ , in the influx.

Groundwater flow is likely to be relatively constant under the TRW property (except where surface features contribute to flow) because the underlying soils are homogeneous. Groundwater levels measured in the monitoring wells and presented

TABLE III.1  
TYPICAL PARTITION COEFFICIENTS AND PERMEABILITIES FOR  
VARIOUS MATERIALS PRESENT ON THE TRW SITE

Material	$K^a$	Saturation Concentration <sup>b</sup> (ppm)	Permeabilities (cm/sec)
Pond sediments (high organic content)	$10^4$ - $10^5$	500-5,000	$10^{-5}$ c
Clay	$10^2$ - $10^3$	5-50	$10^{-6}$ - $10^{-8}$ c
Sandy loam topsoil soil (low organic content)	$10^3$ - $10^4$	50-500	$2.8 \times 10^{-3}$ d
Sand and gravel (depth >5 feet)	$1$ - $10^3$	0.05-50	$8.5 \times 10^{-3}$ d
Wax g	$10^6$	50,000	$<10^{-8}$
Concrete			$10^{-6}$ e

<sup>a</sup>K is the dimensionless ratio between Aroclor 1254 concentration in water and soil material by weight at equilibrium. Values are extrapolated from available data for Aroclor 1242 and other sources. Adjustments were made for particle size and organic content in deriving the values shown.

<sup>b</sup>The saturation concentration is derived by multiplying the saturation concentration of Aroclor 1254 in water (50 ppb) by the appropriate partition coefficient.

<sup>c</sup>Based on typical values for such materials (Bear 1979)

<sup>d</sup>Derived from a soil survey of Stark County, Ohio (USDA 1971)

<sup>e</sup>Estimated

in Table II.2 indicate that groundwater generally flows to the south. Deriving the dimensionless pressure drop,  $I$ , from the well water levels and using the permeability data presented in Table III.1, an average linear groundwater flow rate of  $3.2 \times 10^{-2}$  feet/day (11.7 feet/year) is derived by multiplying the permeability by the driving force. A volume flow rate through a defined vertical area can also be derived for any such area using equation 1. Parameters for groundwater plume models were chosen accordingly.

Hypothetically, a groundwater plume caused by contamination at the Minerva site could lead to human exposure in three ways. Sandy Creek lies 700 feet downgradient of the site and might intercept a potential groundwater plume. Because Sandy Creek traverses residential neighborhoods and a park in Minerva, it may be used for recreational purposes. To be conservative, however, exposure is estimated as if Sandy Creek is a drinking water source. The municipal well in Minerva is 4,000 feet southwest of the TRW plant. A calculated cone of depression due to drawdown from this well is large enough to intercept a plume of groundwater contamination from the site if a plume in fact develops.<sup>1</sup> Finally, personal wells (which tend to be low water volume wells) may lie in the path of a potential plume. (A recent survey indicated that no personal wells are currently located in this area.)

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<sup>1</sup>Yaron Sternberg, May 19, 1983; personal communication

## b. Volatilization

Although PCBs are generally considered nonvolatile compounds because of their low vapor pressure, under certain circumstances significant airborne concentrations of PCBs have been observed (NIOSH 1977). Further, under certain conditions PCBs and other nonvolatile compounds have been shown to volatilize at significant rates (see, for example, Tofflemire and Shen 1979, Hartley 1969, Mackay and Leinonen 1975).<sup>1</sup> Thus, volatilization must be considered as a possible exposure path for PCBs. Volatilization of PCBs can potentially cause contamination of the local atmosphere in the vicinity of contaminated regions at the site. Transport via volatilization can be modeled using the classical two resistance theory of transport across an interface (Thibodeaux 1979) to derive a volatilization rate,  $G$ . The volatilization rate,  $G$ , is a function of a pressure gradient driving force,  $(P^*-P)$ , as depicted in equation 2.<sup>2</sup> The constant of proportionality,  $K_O$ , is a mass transfer coefficient and can be determined from the physicochemical properties of the contaminant and the characteristics of the surrounding medium.

$$G = \frac{MK_O(P^*-P)}{RT} \quad (2)$$

Levels of exposure can then be derived by inputting  $G$  into various available air pollution plume models.

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<sup>1</sup>In many of these cases, suspension of PCB-containing particulates has also been shown to contribute to airborne contamination.

<sup>2</sup>As a worst case, the vapor pressure of a potential contaminant can be substituted for the driving force.



Another important mechanism causing airborne contamination is the generation of airborne particulates contaminated with PCBs. This mechanism is difficult to quantify but has been considered in a qualitative manner. Wind is obviously an important factor. A mitigating factor is that the area is grassy and stable.

Volatilization at the site occurs from two types of sources: open water and contaminated soil. PCB-contaminated oil is not evident on the surface. Generation rates from open water can be calculated using equation 2. Volatilization rates from soil differ from the generation rate in open water by differences between soil and water in their relative affinity for PCBs and in their relative surface area. Most of the surface area of each contaminated soil particle contributing to volatilization faces the interior of a pore space within the soil. Thus, the effective generation rate of airborne contamination from soil is reduced by the tortuous diffusion path that vapors must traverse before they escape into the atmosphere. Models used to account for such behavior are discussed in Thibodeaux (1979). Evapotranspiration is not expected to enhance PCB volatilization significantly (Hartley 1969).

#### c. Surface Runoff

Particulates containing residual concentrations of PCBs can be moved by overland flow during heavy rains. Aside from causing rearrangement of local residuals on site, particulates may be transported to, and deposited in, surface water channels

causing contamination of surface waters and their sediments. Exposure depends on the ultimate uses of the potentially contaminated surface water. Modeling of such processes is extremely complicated; thus, surface runoff will be considered only in a qualitative manner.

## 2. Exposure Evaluation

To derive meaningful exposure estimates from environmental migration models, we considered both a probable case and a worst case. For the probable case, available information about the site was used to provide probable values of input parameters for the transport models employed in calculating exposure levels as a function of distance from each source. Since exposure via certain pathways will occur only after extended periods of time, PCB migration was modeled for time periods sufficient to derive an upper limit on the maximum level of exposure likely to occur at any time in the future. For the worst case, available information about the site was used, but more conservative (worst-possible case) values were chosen so that, subject to the limitations of the models, calculated exposure levels would likely be significantly higher than levels that may actually occur. Although the margins of safety built into the worst case are significantly larger, even the probable case includes considerable margins of safety built into the input values. For example, exposure through surface waters was assumed in both cases to be through drinking water use even though local surface waters are not used for drinking water. Moreover,

worker exposure though air contamination assumes outdoor worker exposure for 40 hours per week for 70 years, an unlikely situation in several respects.

Results from each of the three exposure pathways are presented in the following sections.

a. Exposure via Percolation to Groundwater

Contributions to groundwater contamination due to percolation from each of the regions containing residual concentrations of PCBs on the site, which act as point sources, are considered in the following sections. Briefly, available data from each region were used to determine an effective source strength as represented by the rate of infusion,  $Q$ , and the source concentration,  $C_0$ . In this section, the differences between probable cases and worst cases arise primarily in the treatment of adsorption and permeability. For adsorption, only reversible processes were considered in the worst-case scenarios. Permeability is also treated conservatively in such scenarios. In a final section, infusion rates and source concentrations are compared with groundwater plume modeling results to predict the levels of contamination that may occur in the Minerva well, Sandy Creek, and downgradient personal wells. Levels of contamination are derived by considering the superposition of contributions from each source.

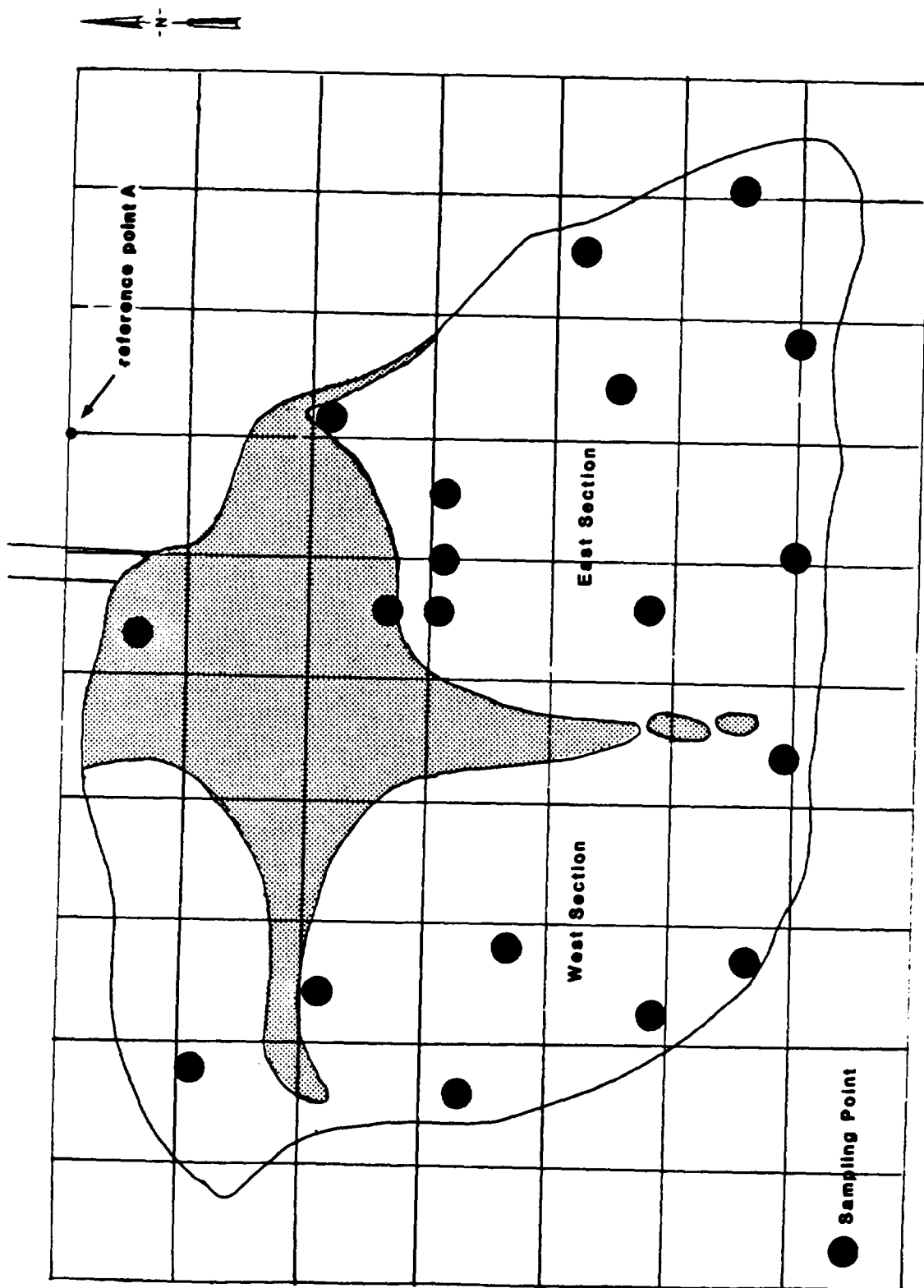
Vertical percolation can be evaluated using equation 1, just as horizontal percolation is. Cases considered for this site fall into two categories. If standing water was present,

the driving force was determined by dividing the height of the standing water (pressure head) by the thickness of the least permeable layer that would have to be traversed to reach groundwater. In the absence of standing water, percolation was estimated from local climate data.

The subsections describing percolation for each potential source contain a summary of relevant source characteristics followed by a description of methods used to derive potential source concentrations and infiltration rates. These are presented in tabular form at the end of each subsection.

#### i. South Pond

South Pond covers an area of 91,000 square feet. Because a large central peninsula and several other exposed areas are situated in the pond (Figure III.2), the water surface area in South Pond is 70,000 square feet. The east and west sections of the pond, separated by the central peninsula, differ markedly in character, suggesting that the pond may have been constructed in stages over an extended period of time. The average depth of the 40,000-square-foot east section is 6 feet. This section contains an average of 1.5 feet of sediment and is relatively free of debris. In contrast, broken concrete, waxy material, and other debris are scattered throughout the 30,000-square-foot west section. Sediment in this section is only 0.5 feet deep on average. The average water depth in the west section is 2 feet, so that the average depth in the entire pond is 4.5 feet. The sampling team at Alert Laboratories reported



50 feet 1 inch

FIGURE III.2

SOUTH POND

that clay was encountered at every core-sampling site in South Pond. Since there is no evidence that natural clay occurs anywhere else on the site, the clay under South Pond is believed to be artificial and intentional. Therefore, this man-made lining is considered in evaluating the percolation rate from South Pond. The source concentration,  $C_0$ , and rate of influx,  $Q$ , can be estimated based on these data and the analytical data on PCB contamination in South Pond presented in the text. Both worst-case and probable-case values are considered below.

For the South Pond, the source concentration,  $C_0$ , is taken to be the concentration of PCBs in the pond water after it percolates through the pond sediments and clay liner. Since groundwater reaches the bottom of the clay liner during the wettest months of the year, it is assumed that PCB concentrations in pond water percolating from the bottom of the liner will remain constant until this water enters the groundwater, if such migration occurs.

For the worst case, it is assumed that the initial concentration of PCBs in the pond water (7 ppb) remains unchanged by interaction with the sediments or clay liner during percolation. This is based on the assumption that the current PCB concentration measured in the pond represents an equilibrium concentration since the water is in continual contact with pond sediments. Thus, PCB concentrations would not change as the water percolates through the sediment. This assumption is believed to be very conservative because deeper sediments have been shown to contain

significantly lower concentrations of PCBs than shallower sediments so that adsorption should occur. The clay liner is also expected to exhibit a substantial adsorptive capacity. It is thus likely that PCB concentrations in percolating water would decrease as the sediments and liner are traversed due to adsorption. These processes are accounted for in the probable-case scenario.

It is assumed for the probable case that interactions (principally irreversible adsorption) with pond sediments and the clay lining serve to attenuate PCB concentrations in the water as it percolates to groundwater. It is assumed that a 70% loss of PCBs occurs due to irreversible adsorption (Di Toro and Horzempa 1982). Thus, each time pond water percolates through a strata of sufficient total capacity, the concentration of PCBs in the water is reduced by 70%. Pond water traverses two such strata: the deeper pond sediments<sup>1</sup> and the clay lining. Thus, if percolation occurs, water entering the groundwater from South Pond would be reduced to approximately 600 ppt ( $0.3 \times 0.3 \times 7$  ppb), which is the value for the probable case.

The rate of water infiltration,  $Q$ , through the pond bottom can also be estimated. The clay lining under South Pond restricts the rate of percolation to groundwater. For the worst case, it is assumed that South Pond has a maximum water depth of

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<sup>1</sup>Results of the sampling program indicate that deeper sediments contain substantially lower concentrations of PCBs than shallower sediments.

6 feet and that the permeability of the clay liner is  $1 \times 10^{-6}$  cm/second, the highest expected permeability for such material (see Table III.1). By use of these assumptions and equation 1, Q for the worst case is calculated to be 1,200 cubic feet/day. For the probable case, it is assumed that the South Pond water depth is 4.5 feet (the reported average depth of the entire pond) and that the permeability of the clay liner is  $1 \times 10^{-7}$  cm/second, the average permeability for such materials (see Table III.1). By use of these assumptions and equation 1, Q for the probable case is calculated to be 89 cubic feet per day.

Results from the foregoing discussion and the calculated total quantities of PCBs injected daily into the groundwater for the most probable and worst cases are as follows:

	$C_o$	Q	Quantity of PCB Injected Daily
Worst case	7 ppb	$1.2 \times 10^3$ ft <sup>3</sup> /day	$5.2 \times 10^{-4}$ lbs/day (0.24 g/day)
Probable case	600 ppt	89 ft <sup>3</sup> /day	$3.3 \times 10^{-6}$ lbs/day ( $1.5 \times 10^{-3}$ g/day)

Note: These estimates can be improved when the pond is drained and the lining is examined in detail.

#### ii. The Swale

The swale is 75,000 square feet in area. It begins at the northeast corner of the plant and runs south for 650 feet before turning west at the southeast corner of the plant. From this point, the swale runs west for 900 feet under two



crossovers and the wax ditch and terminates at the stream emanating from West Lake.

Potential contributions to groundwater contamination from residual PCB concentrations in the swale must be modeled in two ways. Since the majority of residual PCBs in the swale reside in the upper 2 feet, a percolation model is sufficient to account for potential transport of residuals over most of the swale. In the corner section, however, there are two regions where PCB residuals exist at a sufficient depth that, during the wettest months, PCB residuals may come into direct contact with groundwater. These will be modeled separately.

Since there is no standing water in the swale, percolation occurs principally during precipitation. Thus, in order to determine the percolation rate, it is first necessary to determine the fraction of the year that the swale will contain runoff and the average depth of such runoff. To do this, the quantity of water carried by the swale must be determined. The swale drains an area of 150,000 square feet, which includes the cement pad. In heavy rains (0.5 inches per hour), the swale collects 6,250 cubic feet/hour or 1.7 cubic feet/second of runoff from this drainage area.

Water drained by the swale will exhibit a linear flow rate,  $F$ , determined by the slope of the swale. Since the swale has a shallow slope over most of its length, it will drain at 1.5 ft/second.<sup>1</sup> The depth of flow is a function of the

linear flow rate, the volume flow rate, R (1.7 cubic feet/second), and the width, W, of the deepest section of the swale:

$$\text{depth} = \frac{R}{WF}$$

An average width of 10 feet thus yields a depth of 0.11 feet. The percolation rate can now be determined by considering the thickness and permeability of the least permeable layer.

For the worst case, it is assumed that the swale is lined with sediment 1 inch thick, having a permeability of  $1 \times 10^{-4}$  cm/second. This is believed to be a conservative assumption because fine sediments washed from the area drained are likely to collect in the swale. Average depths of such sediments are frequently greater than 1 inch, and permeabilities are frequently lower.<sup>2</sup> The percolation rate derived from equation 1, using these assumptions, must be reduced, however, to account for the fact that such severe storms occur fewer than 20 times per year (NOAA 1982). The resulting worst-case percolation rate is 29 cubic feet/day.

A conservative estimate of the partition coefficient for sediments is  $1 \times 10^4$  (Table III.1). Thus, since typical residual PCB concentrations in the upper layer to the swale are on the order of 300 ppm, a worst-case estimate for PCB concentrations in water percolating through the upper layers of the swale

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<sup>1</sup>Yaron Sternberg, private communication, 1983. This estimate is based on established principles of open channel hydraulics.

<sup>2</sup>Yaron Sternberg, private communication

is 30 ppb. This represents a conservative estimate for the source strength,  $C_0$ , of the swale. Residual PCB concentrations decrease with depth, so water percolating through the deeper zones would be likely to lose PCBs due to adsorption. A probable-case percolation rate is derived by assuming an average permeability for sediment of  $5 \times 10^{-4}$  cm/second and assuming that the sediment depth averages 2 inches. In addition, the assumption that 20 storms per year will yield 0.5 inches of rain for 24 hours is extremely conservative. Five such storms total 60 inches per year, which exceeds the average yearly precipitation rate in the area. Based on these assumptions, and assuming five events per year, a probable-case percolation rate of 7.3 cubic feet/day is derived using equation 1. To ensure that the probable case remains conservative, the same  $C_0$  as derived for the worst case is assumed for the probable case.

Finally, contributions from the deep sections of the corner must be considered. Due to the irregular distribution of residual PCBs in this area, the most likely scenario is that PCBs at depths greater than a few feet in this area are adsorbed on sediment particles that have settled through the coarser particles of the surrounding soil matrix. Since this process favors finer sediments, the partition coefficient of such materials is likely to be  $1 \times 10^{-5}$  cm/second. A conservative estimate of concentrations for these materials is 100 ppm, since the average of 13 deep samples in this area is 10 ppm and presumably includes surrounding soil containing less residual PCBs. Thus

water coming in contact with such material might conservatively retain a PCB concentration of 1 ppb. Therefore, a worst-case source strength,  $C_o$ , for contributions from the deeper corner is 1 ppb. There are two areas in the deep corner where residual PCBs appear to be more concentrated than other deep areas. A conservative estimate of their combined cross section is 200 feet by 5 feet. Thus, the two together represent a total cross section of 1,000 square feet. Multiplying this area by the linear groundwater velocity ( $3.2 \times 10^{-2}$  feet/day) yields a worst-case percolation rate,  $Q$ , of 32 cubic feet/day for contributions from the deep corner. As a conservative measure, the probable-case values for contributions from the deep corner are assumed to be the same as the worst case. Worst-case and probable-case source strengths, percolation rates and injection rates calculated for the swale are summarized below. It should be noted that deep corner contributions are believed to be insignificant compared to percolation.

	$C_o$	$Q$	Quantity of PCB Injected Daily
<u>Worst Case</u>			
Percolation contribution	30 ppb	29 ft <sup>3</sup> /day	$5.4 \times 10^{-5}$ lbs/day
Deep corner contribution	1.3 ppb	32 ft <sup>3</sup> /day	$2.6 \times 10^{-6}$ lbs/day
Total			$5.6 \times 10^{-5}$ lbs/day
<u>Probable Case</u>			
Percolation contribution	30 ppb	7.3 ft <sup>3</sup> /day	$1.4 \times 10^{-5}$ lbs/day
Deep corner contribution	1.3 ppb	32 ft <sup>3</sup> /day	$2.6 \times 10^{-6}$ lbs/day
Total			$1.6 \times 10^{-5}$ lbs/day

### iii. The Rubble Pile

Since there is no standing water on the rubble pile, potential contributions to groundwater are driven by precipitation. For a worst case, we assume that 30 inches/year of rain will percolate through the rubble pile. Since the total yearly precipitation in this area averages 40 inches per year (NOAA 1982), the assumption that 75% will percolate rather than run off or evaporate, represents an extremely conservative assumption. Since the rubble pile covers an area of  $2.3 \times 10^4$  square feet, this represents a potential percolation rate,  $Q$ , of 160 cubic feet/day as a worst case. A more reasonable assumption is that 25% of total precipitation or 10 inches/year will percolate through the rubble pile. Thus, the probable-case percolation rate,  $Q$ , for the rubble pile is 53 cubic feet/day. This also represents an extremely conservative assumption because residual concentrations are patchy, so that only a portion of the percolating water may actually come into contact with residual PCBs.

Average residual PCB concentrations in the rubble pile are on the order of 100 ppm. The higher concentrations tend to be associated with organic residues (waxes) in the pile so that a partition coefficient on the order of  $1 \times 10^6$  is likely. Thus, a worst-case source strength,  $C_0$ , of 100 ppt is derived from the above assumptions. This value is also used for the probable case.

These values of  $C_0$  and the resulting PCB injection rates are summarized below.

	Co	Q	Quantity of PCB Injected Daily
Worst case	100 ppt	160 ft <sup>3</sup> /day	9.9x10 <sup>-7</sup> lbs/day
Probable case	100 ppt	53 ft <sup>3</sup> /day	3.3x10 <sup>-7</sup> lbs/day

#### iv. The Wax Ditch

The permeability of the residue in the wax ditch is expected to be very low: less than 10<sup>-8</sup> cm/second. Coupled with a small total area, percolation from the wax ditch is insignificant compared to contributions from other areas on the site.

#### v. Fry Property

Although localized areas of residual PCB concentrations exist, the total surface area occupied by these spots and the total quantity of PCBs on the Fry property are minimal (Table II.1).

#### vi. Total

Hypothetical levels of exposure due to groundwater contamination from each source were obtained for the Minerva well, Sandy Creek, and personal wells in a similar manner. Using South Pond as an example, the method is outlined in the following paragraphs.

The Minerva well withdrawal rate is 750 gallons/minute (1.4x10<sup>5</sup> cubic feet/day). Assuming all of the influx from South Pond, 0.24 grams/day at worst, would be intercepted by

the Minerva well, the PCB concentration in the well would not exceed 60 ppt. Further, this hypothetical level of contamination would not be reached for 350 years. Using the probable-case values for South Pond, the maximum concentration to be observed in the Minerva well would be 0.38 ppt and would not occur for 350 years.

In order to evaluate potential contributions to Sandy Creek, dispersion must be considered. Based on groundwater modeling results, a conservative assumption appears to be that a potential contaminant plume will disperse at one-tenth the rate of flow. Therefore, since the local aquifer is 90 feet deep, a potential plume will disperse over the entire thickness of the aquifer by the time it travels 900 feet downgradient. Since the closest potential source on the TRW site is on the order of 900 feet from Sandy Creek, it is expected that any potential plume from these sources would be dispersed over the entire thickness of the aquifer by the time Sandy Creek is reached.

The flow rate in Sandy Creek was estimated at  $1.1 \times 10^6$  cubic feet/day on December 14, 1982. It is assumed that during dry periods, the flow rate would be 10% of the rate observed. However, Sandy Creek is no more than 6 feet deep, and the aquifer is 90 feet deep. Thus, even assuming conservatively that groundwater flow is modified in the vicinity of the creek so that the first 10 feet of aquifer will be intercepted, only 10% of a potential contaminant plume will contribute to PCB contami-

nation in Sandy Creek. Thus, potential concentrations in Sandy Creek can be derived by dividing 10% of the quantity of PCBs injected daily from a source by the flow in Sandy Creek. To be conservative, the low flow case is assumed.

Hypothetical levels of contamination predicted in future personal wells due to each source were also obtained by considering dispersion. Such wells would probably be located south of Sandy Creek, if at all. It was therefore assumed that potential plumes would travel at least 1,000 feet before being intercepted by any personal wells and that dispersion would result in plume concentrations smaller than the initial source strength,  $C_0$ . Dispersion was assumed to occur both transversely and vertically. Transverse dispersion was assumed to add 200 feet (100 feet on each edge) to the width of the potential plume, which has an initial width equal to the width of the source perpendicular to the direction of groundwater flow. Vertical dispersion results in a plume depth of 90 feet. Hypothetical well concentrations were thus derived from the daily quantity of PCBs injected by a potential source divided by the volume flow across a disperse area in the groundwater with dimensions of 90 feet by 200 feet plus the transverse width of the potential source. For example, South Pond stretches 350 feet across the direction of groundwater flow. After 1,000 feet, a potential plume from South Pond occupies an area of 90 feet by 350 plus 200 feet or  $5.0 \times 10^4$  square feet. The volume flow is obtained by multiplying this area by the linear flow rate of groundwater,



$3.2 \times 10^{-2}$  feet/day, to yield  $1.6 \times 10^3$  cubic feet/day. The worst-case concentration in such a hypothetical plume is derived by dividing the worst-case injection rate by the weight equivalent of the volume flow in the plume at the point of interception. Thus, at worst, a hypothetical plume from South Pond would yield a concentration in personal wells of 5 ppb.

Calculated levels of contamination predicted from all sources for the Minerva well, Sandy Creek, and potential future personal wells are summarized in Table III.2. Hypothetical lifetime average daily doses for individuals who might drink such water are also presented. (Lifetime average doses were derived assuming that contaminated sources are used for drinking and that the average individual consumes 2 liters a day.)

b. Exposure via Volatilization

Contributions to airborne contamination from each source were determined in two steps. First a generation rate was derived for each source based on two factors: the total surface area of the region containing residual PCBs and the average concentration of residual PCBs. (Most of these data were presented in earlier sections of this document.) As noted earlier, water surfaces were modeled using equation 2, and soil surfaces were modeled by adjusting this equation to account for diffusion through soil pore spaces (Thibodeaux 1979).

Airborne concentrations downwind of each source were determined by inputting derived generation rates into an available desk top dispersion model (Turner 1970). Contaminant concentrations at the source were estimated by assuming that turbulence

TABLE III.2  
POTENTIAL EXPOSURE DUE TO  
PERCOLATION FROM SOURCES AT THE TRW SITE<sup>a</sup>

Source	Level of Exposure <sup>b</sup>		
	Minerva Well	Sandy Creek	Personal Wells
South Pond	0.38 ppt (60 ppt)	0.03 ppt (6 ppt)	33 ppt (5 ppb)
Swale	6 ppt (6 ppt)	0.6 ppt (0.6 ppt)	370 ppt (3 ppb)
Wax Ditch	-----Insignificant-----		
Rubble Pile	-----Small-----		
Fry Property	-----Insignificant-----		
Total <sup>c</sup>	6 ppt (65 ppt)	0.6 ppt (7 ppt)	370 ppt (5 ppb)
Drinking Water Dose Per Person Assuming Plumes Develop (µg/day)			
Total <sup>c</sup>	0.012 (0.12)	<0.01 (0.014)	0.74 (10)

<sup>a</sup>No exposure will occur for at least 100 years, if at all.

<sup>b</sup>Results are presented in the following format: probable case (worst case).

<sup>c</sup>Superposition varies by method of calculating exposure. It is not necessarily additive.

causes a volume over the source to be well mixed to a height of 2 meters and that this volume is renewed by local wind. Results of these calculations are presented in Table III.3. (Doses are calculated assuming that an average individual breathes at 10 liters/minute and that all PCBs entering the lungs are adsorbed.)

c. Exposure via Surface Runoff

As detailed earlier, this mechanism is potentially important but nearly impossible to quantify. Therefore, this route of migration is treated only qualitatively. Effects are summarized in Table III-4.

B. Toxicity Assessment

The toxicity of PCBs is evaluated in this section, which includes a survey of the literature, consideration of the difficulties of such evaluations, a review of key studies, and a discussion of limitations to such studies. Margins of safety are considered in the final segment of this section.

1. Literature Survey

Scientific data on the toxicity of PCBs and of their contaminants and related compounds have been reviewed in many papers, books, and scientific reports. Appendix C lists 25 of these scientific reviews published between 1972 and 1983. We have reviewed most of the available scientific information summarized in these reviews and published in recent scientific journals. The scientific literature on the toxicity of PCBs is voluminous and complex, and a wide variety of toxic effects

TABLE III.3

## HYPOTHETICAL EXPOSURE DUE TO VOLATILIZATION FROM SOURCES AT TRW SITE

Source	Breathing Dose Per Person Downwind ( $\mu\text{g}/\text{day}$ ) <sup>a</sup>				
	At Source	100 m Downwind	500 m Downwind	1,000 m Downwind	10,000 m Downwind
South Pond	$3.6 \times 10^{-3b}$ (0.20)	$0.66^c$ (2.8)	$0.14^d$ (0.14)	$4.2 \times 10^{-2}$ ( $4.2 \times 10^{-2}$ )	$6.5 \times 10^{-4}$ ( $6.5 \times 10^{-4}$ )
Swale	Small	$0.54^c$	$2.8 \times 10^{-2}$	$7.3 \times 10^{-3d}$	$1.2 \times 10^{-4}$
Wax Ditch	-----Insignificant-----				
Rubble Pile	Small	0.09 (0.24)	$4.9 \times 10^{-3d}$ ( $1.4 \times 10^{-2}$ )	$1.3 \times 10^{-3}$ ( $3.3 \times 10^{-3}$ )	$2.1 \times 10^{-5}$ ( $5.4 \times 10^{-5}$ )
Fry Property	-----Insignificant-----				
Total <sup>e</sup>	$3.6 \times 10^{-3}$ (0.2)	$1.0^c$ (2.8)	$0.1^d$ (1.4)	$4.2 \times 10^{-2}$ ( $4.2 \times 10^{-2}$ )	$7.8 \times 10^{-4}$ ( $8.1 \times 10^{-4}$ )

<sup>a</sup>Results are presented in the following format: probable case, (worst case) except for the swale where the two cases are identical.

<sup>b</sup>Assumes no one remains by pond for more than working 20 days/year.

<sup>c</sup>Since these represent "on site" exposures, assumes no workers are exposed for more than 2,080 hours/year.

<sup>d</sup>This is first distance where local inhabitants are likely to be exposed, so represents continual exposure.

<sup>e</sup>Superposition is a complicated function that is not directly additive. Numbers were derived by inspection.

TABLE III.4  
POTENTIAL MIGRATION DUE TO SURFACE RUNOFF

Source	Exposure Potential	Point of Contribution	Probable Effect
South Pond	Small	Fry property, Sandy Creek	Occurs only during flooding. May have caused contamination on Fry property. Effect is now probably small because pond has stabilized and new water is not being introduced except for precipitation.
Swale	Medium*	Sandy Creek	Surface runoff from swale might traverse stream emanating from West Lake and reach Sandy Creek.
Wax ditch	Small	South Pond	Movement from wax ditch to South Pond is hindered by debris.
Rubble pile	Medium	Neighboring property	The rubble pile slopes down toward neighboring property.
Fry property	Small	Sandy Creek	The total quantity of PCBs on the Fry property is small and a large area would have to be traversed to reach Sandy Creek.

\*Sampling has revealed no evidence that such contamination has occurred. However, sampling has not been performed immediately following storm events, when such migration is most likely to occur.

has been documented in studies of varying quality and conclusiveness. Appendix D to this report summarizes the toxicity studies which we judge to be the most relevant to assessing the possible hazards posed by the current contamination of the Minerva site with PCBS.

Data on effects of PCBs in humans are derived from three main sources.

- In studies of workers exposed to relatively high levels<sup>1</sup> of PCBs in the workplace, the most consistent effect reported is a skin disease known as chloracne. Other effects reported less consistently or less frequently include induction of hepatic microsomal enzymes, other changes in liver function, neurological and behavioral symptoms, digestive disturbances, eye irritation, and respiratory impairment (NIOSH 1977, Warshaw et al. 1979). Three studies have suggested possible excess frequencies of cancer in exposed workers (Bahn et al. 1976, Brown and Jones 1981, Bertazzi et al. 1981), but these studies were limited by small sample sizes, were mutually inconsistent, and are not generally regarded as conclusive.
- Two incidents have been reported in which large numbers of people were poisoned by consumption of cooking oil contaminated with PCBs and with their thermal decomposition products. These incidents took place in Japan, where the disease is known as "Yusho" (Kuratsune et al. 1972, 1976), and in Taiwan (Chang et al. 1980). The symptoms included long-lasting skin lesions similar to those observed in chloracne, but also included abnormal pigmentation of the skin, menstrual disorders, liver damage, neurobehavioral impairment, minor birth abnormalities, respiratory symptoms, and immunological impairment.
- In the general population, exposure to PCBs (as measured indirectly by residues of PCBs in the blood or body fat) has been statistically correlated with elevated blood pressure (Kreiss et al. 1981), elevated levels of liver enzymes and serum triglycerides (Baker et al. 1980), premature births (Wassermann et al. 1982), missed abortions (Bercovici et al. 1980), and various types of cancer (Unger and Olsen 1980). However, these effects

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<sup>1</sup>"High" occupational levels are 200-2000  $\mu\text{g}/\text{m}^3$  plus skin contact.

were also associated with exposure to DDE and other chemicals, and the results are ambiguous.

Experimental studies in animals have revealed a wide spectrum of adverse effects of PCBs when administered under appropriate conditions. Some of these effects are closely parallel to effects observed in humans. For example, PCBs induce hepatic microsomal enzymes in a number of animal species, and cause skin lesions in rhesus monkeys that closely resemble those observed in human chloracne. Some effects observed in animals, such as immunosuppression and gastric lesions, have been reported only infrequently or equivocally in humans. Other effects have not been observed in humans, such as the induction of birth defects in dogs and swine. Several studies have indicated that PCBs can cause or enhance the induction of liver cancer in rats and mice.

## 2. Problems in Hazard Assessment

Assessment of the hazards posed by PCBs is made very complex by a number of factors.

- PCBs are complex mixtures and a number of commercial products have been sold and used which varied substantially in composition. It has long been known that these products differ in toxicity to at least a moderate degree. Some of the most serious toxic effects in animals (e.g., reproductive impairment in rhesus monkeys and liver cancer in rats) have been demonstrated only in experiments with mixtures (Aroclors 1248 and 1260) that were never used commercially in large quantities.
- PCBs are subject to contamination by highly toxic impurities, of which the most important are polychlorinated dibenzofurans (CDFs). These impurities were present at only trace quantities (1-5 ppm) in the commercially pure PCB mixtures, but can be formed in service under certain conditions involving high temperature and the

presence of oxygen. There is little doubt that the high toxicity of the PCB mixtures in the "Yusho" incidents in Japan and Taiwan was due primarily to CDFs formed in service in this way.

- The results of studies with PCBs, especially in exposed human populations, have often been inconsistent, and some positive results have not been confirmed when the studies were repeated under similar conditions.
- Some studies have been of poor quality and can be accorded little weight in hazard assessment.
- The effects of PCBs often appear widely different in different species of animals. For example, species such as the mink and the rhesus monkey appear to be extremely sensitive to certain PCB mixtures, whereas the more conventional test species such as rats, mice, and dogs appear much less sensitive.
- The human studies have been limited in scope and duration, and they have not yielded conclusive evidence about the potential of PCBs to cause reproductive impairments or cancer in exposed populations.

For these reasons, selection of scientific studies to serve as the basis for risk assessment for PCBs and CDFs is unusually difficult. On the one hand, it can be argued that studies in animals provide evidence for serious toxic effects at very low exposure levels. In particular, the toxic effects observed in rhesus monkeys are parallel to those in humans exposed to PCBs either in the workplace (chloracne) or via contaminated food (Yusho). Studies in rhesus monkeys have revealed serious effects on reproduction and neurobehavioral development at low exposure levels. On the other hand, it can be argued that chloracne and Yusho are probably caused primarily by CDFs, that the monkeys were exposed to a mixture (Aroclor 1248) to which there has been very little human exposure, that reproductive and neurobehavioral effects were not



reported in Yusho victims, and that more conventional test animal species have not shown significant effects at low exposure levels. With reference to human studies, it can be argued that studies of workers exposed to PCB mixtures made in the United States have not unequivocally shown significant effects, except for chloracne at high exposure levels and inconsistent effects on liver function.

These arguments have been used by Ecology and Environment (1981) and Drill et al. (1982) to support the conclusion that risks posed by PCBs at low levels in the environment are insignificant. However, this conclusion has not yet been adopted as a consensus view by other scientific reviewers (see Appendix C) or by regulatory agencies such as the U.S. Environmental Protection Agency (USEPA 1980). Pending resolution of this scientific debate, for the purposes of this risk assessment we place weight on the scientific studies that suggest that PCBs may be toxic at low exposure levels. However, we recognize that the relevance of these studies has been questioned and that our assessment may be unduly conservative.

To serve as the basis of our risk assessment, we have selected three types of toxic effect of PCBs that are judged most likely to be relevant at low exposure levels. (Appendix D explains why other types of toxic effect are considered less relevant, and why exposure to CDFs at Minerva is unlikely.) The following section summarizes data on these toxic effects, and assesses their importance at low dose levels.

### 3. Assessment of Key Studies

#### a. Chloracne

Chloracne is a disfiguring skin disease that has been associated with exposure to PCBs, both in the workplace (NIOSH 1977) and through accidental contamination of food (Kuratsune 1972, 1975; Chung et al. 1980). The incidents of food contamination are complicated by the presence of high levels of CDFs and other contaminants, so the following assessment is based on dose-response data obtained through studies of workplace exposure to relatively uncontaminated PCBs.

NIOSH (1977) reviewed the data on the occurrence of chloracne in workers exposed to PCBs, and concluded that chloracne was generally associated with PCB concentrations in the blood of 200 ppb or greater. Fischbein et al. (1979) reported a high incidence (45-55%) of skin lesions (not limited to chloracne) in workers in a capacitor manufacturing plant whose average plasma concentration of PCBs was 172 ppb. Because of wide variability in blood concentrations and uncertainty in diagnosis, these and other reports on occupational exposure do not clearly specify a no-observed-effect-level (NOEL) for chloracne. However, Humphrey (1977) reported no significant increase in skin disorders in groups of people exposed to PCBs via fish, with mean blood levels of PCBs ranging from 46 to 82 ppb, and individual levels ranging up to 366 ppb. Recognizing the possibility that chloracne may be caused primarily by CDFs, we will use these data as the basis for the assumption that 80 ppb in the

blood is a NOEL for skin effects of PCBs in healthy adults exposed to environmental residues of PCBs. This figure is selected as the highest average level for a group of people with no overtly detectable effects.

This blood concentration can be converted to equivalent PCB intake, using the results of the study by Humphrey (1977), which indicated that a blood concentration of 58 µg/liter corresponded to an average daily intake of about 130 µg/day. Assuming that the relationship between intake and blood PCB level is linear, an average daily intake of PCBs of about 180 µg ( $80 \times 130/58$ ) would be a NOEL.

b. Reproductive and Neurobehavioral Effects

Allen and his coworkers have reported a series of studies of the effects of PCBs (Aroclor 1248) on the health and reproductive performance of rhesus monkeys (Allen et al. 1975, 1979; Bowman et al. 1978, 1981). At relatively high concentrations in the diet (10 and 5 ppm), PCBs severely impaired reproduction, and the few infants who were born alive showed signs of poisoning, some of which closely resembled the signs of chloracne in man. Similar (but less severe) effects were observed in monkeys exposed to PCBs at 1 and 0.5 ppm on the diet. In the most recent study (Bowman et al. 1981), monkeys exposed to PCBs at 0.5 ppm in the diet for 3 days per week throughout pregnancy and nursing bred more or less normally, but the infants were underweight at birth, developed minor signs of poisoning (hyperpigmentation of skin) during the nursing period, were

hyperactive and showed impaired learning ability when tested at 6 months of age. This average dietary concentration of 0.21 ppm is the lowest dose level at which significant adverse effects of PCBs have been reported in experimental animals. This dietary concentration is equivalent to a daily dose of 0.011 mg/kg body weight/day in the monkeys, which would correspond to a daily intake of about 700 µg/day in adult humans.

c. Carcinogenic Effects in Animals

Several studies have shown that PCBs are carcinogenic in rats and mice, causing increased incidence of liver tumors when administered to the animals at high levels in the diet for most or all of their lifetimes. However, only two of the studies were conducted systematically enough to serve as the basis for risk assessment. In a study by Kimbrough et al. (1975), lifetime exposure of rats to a diet containing 100 ppm Aroclor 1260 led to a high incidence (26/184 or 14.3%) of carcinomas of the liver, and a very high incidence (144/184 or 78.3%) of neoplastic nodules. In a smaller study by the National Cancer Institute (1978), lifetime exposure of rats to a dietary concentration of 100 ppm Aroclor 1254 led to a much smaller incidence of liver tumors (2/48 carcinomas and 3/48 neoplastic nodules or adenomas, pooling data for males and females). Although the differences in tumor frequency between the two experiments are large, the biological difference is less important, because the rats in the NCI study also had a high incidence of "hyperplastic nodules," a condition regarded

by many pathologists as an earlier stage in the development of liver tumors. Thus, the main difference appears to have been that tumors developed more rapidly in the Kimbrough experiment than in the NCI experiment.

Several writers have used the data of Kimbrough et al. (1975) as the basis for low dose risk assessment. The most comprehensive assessment was that of Crump and Masterman (1979), who calculated that a dietary concentration of 6.9 ppb would correspond to a lifetime excess risk of  $10^{-5}$  (1 in 100,000) for hepatocellular carcinomas, and that a dietary concentration of 0.63 ppb would correspond to a lifetime excess risk of  $10^{-5}$  for neoplastic nodules. If it is assumed that humans would respond similarly at the same dietary concentrations, then a lifetime excess risk of  $10^{-5}$  would correspond to daily intakes of about 10 ug and 1 ug, respectively.<sup>1</sup>

It should be emphasized that these estimates of possible carcinogenic risk are tenuous. They involve extrapolation from an effect observed in rats at a very high dose level to predict effects in humans at doses 100,000 times smaller. Although this extrapolation follows EPA's current procedures for carcinogenic risk assessment, these procedures are intended to be conservative, and are currently under review. PCBs are known to act by "promoting" the effects of other carcinogens

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<sup>1</sup>Similar results were presented by NAS (1977) and EPA (1980). The results are sensitive to the assumption that is made about the way in which doses should be scaled between species (Nisbet 1981), but the assumption made in the text conforms to EPA's current practice.

(Nishizumi 1976, 1979, Preston et al. 1981). However, these effects have been studied only at high doses, and there are no accepted procedures for risk assessment in these circumstances. Some recent writers have proposed that thresholds exist for the action of cancer promoters and that safe levels of exposure can be established by applying a margin of safety to a no-observed-effect-level. However, this approach cannot yet be applied to PCBs because no experiment has been conducted to establish a NOEL for cancer promotion.

#### 4. Limitations of the Studies

Table III.5 summarizes the three types of toxic effects that are considered in our risk assessment. The column headed "Comments" indicates that each of the three sets of data has substantial limitations and that there are several reasons to question their direct applicability to prediction of human risks. Nevertheless, they include the best-documented subchronic effect in humans and the two effects of greatest concern that have been reported in experimental animals. For these reasons we use them as the basis for risk assessment, while emphasizing that the limitations pointed out in Table III.5 may lead to overestimation of risks.

#### 5. Margins of Safety

For each of the three types of toxic effect considered in this risk assessment, some information is available on dose-response relationships. That is, for at least one level of dosage, effects have been measured in animals or humans under

conditions of prolonged exposure. Accordingly, for any hypothetical situation in which humans would be exposed for long periods to lower levels of PCBs, "margins of safety" can be calculated.

Figure III.3 presents a graphic description of the relationship between levels of intake and margins of safety. The left-hand column indicates (on a logarithmic scale) daily intakes of PCBs in  $\mu\text{g}/\text{day}$  and indicates the equivalent long-term average intake levels at which toxic effects have been observed. The middle column characterizes the margin of safety at various lower dose levels. For example, an average daily intake of about 100  $\mu\text{g}/\text{day}$  would be slightly greater than half the no-observed-effect level (NOEL) for chloracne, and about one-seventh that caused adverse effects in monkeys. These margins of safety would normally be considered inadequate, because they would not allow sufficiently for variations in sensitivity within the human population, and for differences in sensitivity between human and animal species. On the other hand, an average daily intake of about 1  $\mu\text{g}/\text{day}$  would be about 1/180 times the NOEL for chloracne, and about 1/700 times the lowest observed effect level in monkeys. These margins of safety would ordinarily be considered adequate to ensure safety to exposed populations, even under conditions of long-term exposure. If average daily intakes were kept below 1  $\mu\text{g}/\text{day}$ , the margins of safety for these effects would ordinarily be considered at least ample.

TABLE III.5  
TOXIC EFFECTS CONSIDERED IN RISK ASSESSMENT

Effect	Dose-Response Data	Comments
<u>Choracne</u>		
Occupational skin disease, liver and neurobehavioral symptoms in some cases	Associated with blood levels over 200 ppb; no effects in groups with 46-82 ppb	Reported effects variable; may reflect misdiagnosis and effects of CDFs; not necessarily the most sensitive indicator of long-term exposure to PCBs
<u>Reproductive Impairment</u>		
Failure to conceive, skin lesions, retarded development, learning deficits	Minor effects at lowest dose tested, about 11 µg/kg/day to mothers	Demonstrated only in rhesus monkeys; other species less sensitive Demonstrated only with Aroclor 1248; possible role of CDFs; small sample
<u>Cancer Induction</u>		
Liver cancer in rats in one good and several unsatisfactory experiments; negative or marginal effects in others	Tested only at very high dose, 100 ppm in diet or about 8 mg/kg/day	Demonstrated only with Aroclor 1260; weak or equivocal effects with Aroclor 1254; primarily cancer promoter



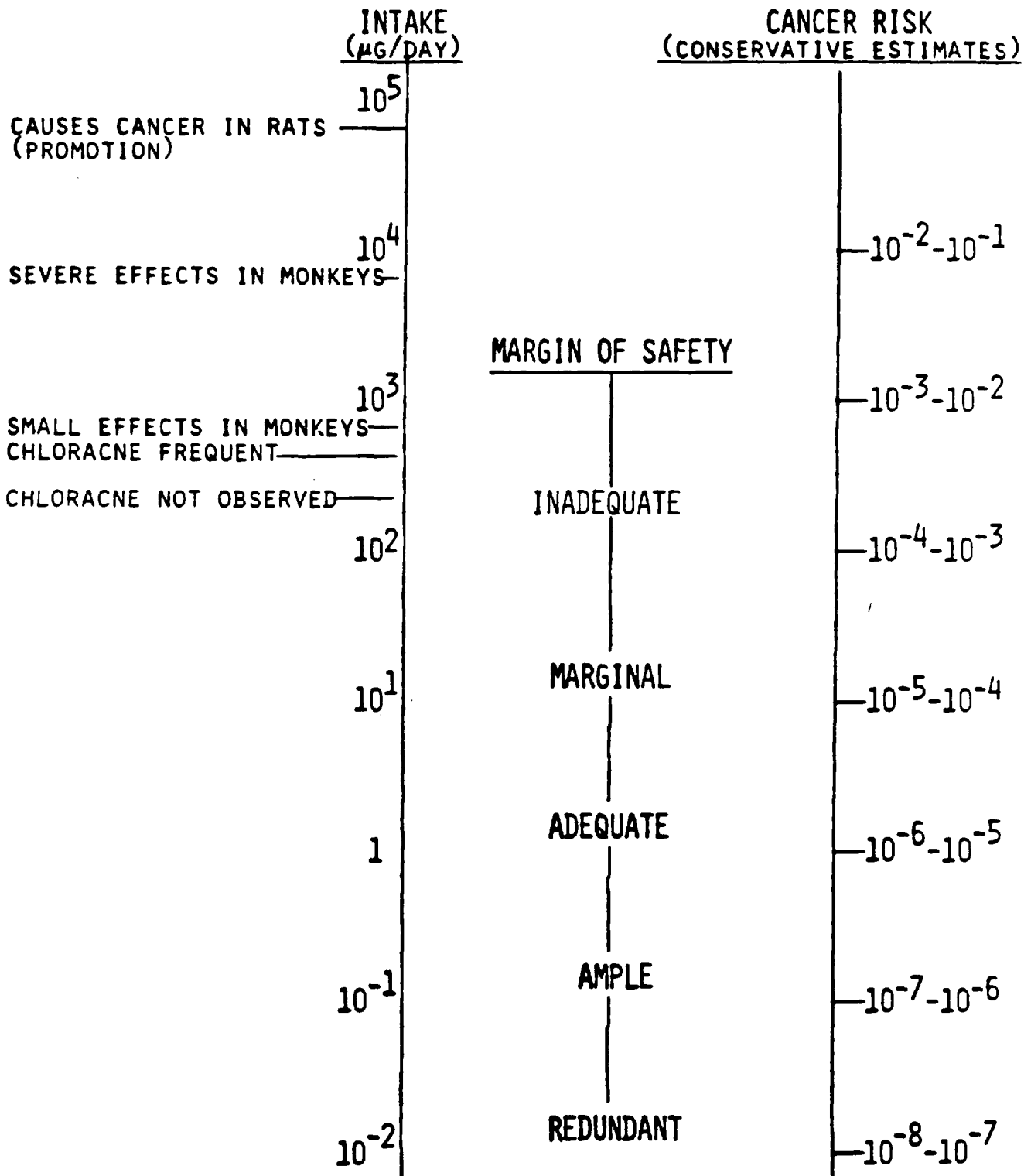


FIGURE III.3

MARGINS OF SAFETY FOR LONG-TERM INTAKE  
OF PCBs AT VARIOUS DAILY DOSE LEVELS

The right hand column in Table III.5 shows conservative estimates of the possible cancer risks that might result from long-term (lifetime) intake of PCBs at the indicated rate. For example, continuous ingestion of 1 µg/day of PCBs throughout life might, under the assumptions of the calculations, lead to cancer risks in the range of  $10^{-6}$  and  $10^{-5}$  (1-10 per million). For the reasons explained above, these estimates are tenuous and are designed to be conservative.

The risk assessments illustrated in Figure III.3 are made specifically for continuous intake of PCBs for long periods (lifetime intake for cancer risks, exposure for months or years for other types of effect). If exposure is for short periods or is intermittent, margins of safety would be correspondingly higher.

### C. Risk Evaluation

Human health risks attendant to residual PCB concentrations as they presently exist at the TRW site, or as they might exist in the future, are evaluated by combining results of exposure and toxicity assessments. It should be emphasized that there is no indication that exposure is actually occurring at present. Predicted current and future levels of exposure were presented for each transport mechanism--percolation, volatilization, and surface runoff in Tables III.2, III.3, and III.4, respectively. In this segment, exposure levels are compared directly with effect levels, which were derived from an evaluation of available toxicological studies, to determine margins of safety

as indicated in Figure III-3. In this manner, conclusions could be drawn about the potential magnitude of risks to people who may be exposed to PCBs via various environmental pathways. Current risks from volatilization are considered first. Potential groundwater contamination and surface runoff represent future risks.

#### 1. Workers On Site

Results of this assessment suggest that inhalation is the only potentially significant exposure pathway for workers on site. Worker practices and other precautions (such as the construction of barrier fences) implemented at the site limit the potential for exposure and attendant risks due to groundwater contamination or surface runoff.

##### a. Volatilization

Table III.3 indicates that intake via inhalation is likely to be less than 1  $\mu\text{g}/\text{day}$  except in all worst-case situations for persons within a few hundred meters. Since there are no residents within 400 meters in the direction of the prevailing wind, which is from the southwest (NOAA 1982), this exposure is of concern only for workers on site. Under worst-case exposure conditions, the average intake of workers who spend a substantial fraction of their working hours outdoors might be as high as 3  $\mu\text{g}/\text{day}$ . This is unlikely, however, because a worker would be required to stand on the pond berm continuously in order to receive such a level of exposure. Under more probable exposure conditions, their average intake would be less

than 1 µg/day (Table III.3). Thus, for a worst-case exposure, the margins of safety are expected to be adequate to prevent toxic effects attributable to PCB exposure. The estimated cancer risk (assuming long-term exposure) would be no higher than  $10^{-5}$ .

b. Groundwater Contamination

Worker exposure to groundwater contamination is expected to be nonexistent.

c. Surface Runoff

Because of protective measures implemented at the site, worker exposure due to surface runoff is expected to be nonexistent.

2. The Minerva Population

Potential risks to the Minerva population derive from exposure due to volatilization, groundwater contamination, and surface runoff. Each of these is considered below.

a. Volatilization

The closest residents to the TRW facility reside 400 meters southeast of the plant. Since local winds seldom blow toward the southeast, average exposure levels will be much lower than those reported in Table III.3. Even under worst-case conditions, levels of exposure to these residents is not likely to exceed 0.2 µg/day representing an ample margin of safety and a cancer risk less than  $10^{-6}$ . Although suspension of contaminated particulates was not considered in this evaluation, risks due to airborne PCB contamination from the TRW site are expected to be insignificant.

b. Groundwater Contamination

The source of PCB contamination exhibited by the groundwater monitoring wells is subject to question at this time. No matter which point source is considered, it is difficult to reconcile the observed pattern of data with results of the groundwater models at present. At the same time, data from filtered samples suggest an alternate source of the observed contamination--suspended sediment from local surface contamination.

The mechanism that is apparently contributing to groundwater contamination in well 10 is vertical sediment migration from local surface soil contamination. Fine particulates contaminated with PCBs from the surface are traversing the pores in the coarser soil material of the local alluvium and migrating into the well. The driving force of this mechanism may either be gravity or water percolation.<sup>1</sup> Based on worst-case estimates of predicted future exposure, prudence dictates that groundwater

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<sup>1</sup>Vertical sediment migration is not expected to contribute significantly to general groundwater contamination because the rate of groundwater flow is insufficient to divert the vertical migration of such materials significantly. Further, desorption from such fine particles is expected to be minimal due to their high affinity for PCBs. In areas where surface contamination is significant, however, this mechanism can contribute to the appearance of contamination in a local well because PCB-containing sediments suspended in well water will contribute to the apparent PCB concentration unless the water is first filtered. Well 10 is located on the Fry property where localized regions of significant surface contamination have been detected. There are two reasons why sediment contamination may not exist in other wells on site to the same degree. First, most wells are not located in areas where significant surface contamination exists. Second, the mechanism is highly irregular and depends on the individual drainage characteristics of regions around each well.

monitoring be continued. The three pathways (via the Minerva Well, Sandy Creek, or personal wells) that are likely to lead to exposure from potential groundwater contamination occurring on site are considered in the following paragraphs.

Table III.3 suggests that if a contaminated groundwater plume were to develop and affect the Minerva well (an event that would potentially occur at least 350 years in the future), the worst-case exposure scenario indicates average daily intakes no greater than 0.12  $\mu\text{g}/\text{day}$ . Such an intake would provide ample margins of safety and would correspond to an incremental cancer risk no greater than  $10^{-6}$ .

There is no evidence now for contamination of the water in Sandy Creek. Contamination of the creek via groundwater would lead to risks an order of magnitude smaller than those of the Minerva well (Table III.2) and are not considered significant. However, there is also a potential for contamination via surface runoff, especially from the swale, to an extent that is difficult to calculate (Table III.4). Human exposure to PCBs via the creek is unlikely to be significant unless the compound is concentrated in fish or aquatic animals.

No personal wells are currently in use in the area that might be affected by a potential groundwater contaminant plume moving away from the site. However, if such a plume were to develop (and were not intercepted by Sandy Creek, as appears likely) and if wells were dug into it (all low probability events), users of such wells might take in PCBs at rates up

to 10 µg/day (Table III.2). These intakes would provide margins of safety that are only marginally sufficient and might correspond to cancer risks in the range between  $10^{-3}$  to  $10^{-4}$ . Although the number of people who might experience such risks is very small (if not zero), it appears desirable at least to monitor for the development of a groundwater plume, and if necessary to conduct remedial measures.

### c. Surface Runoff

The possibility that PCB migration offsite via surface runoff may hypothetically lead to exposure due to contamination of Sandy Creek represents one of the two significant concerns listed in Table III.4. Although no actual sediment contamination has been detected in the stream leading to Sandy Creek, sediment in this stream should be analyzed periodically until the potential for contaminant runoff has been better quantified.

The second significant concern discussed in Table III.4 is the potential for runoff from the rubble pile to spread contamination onto neighboring property. Sampling to date has been insufficient to eliminate this possibility as a concern, and it should be investigated further.

## 3. Conclusions

Results derived from the above risk assessment are summarized in Tables III.6 and III.7. Table III.6 represents worst-case doses calculated in the above exposure assessment coupled with corresponding margins of safety derived in the

TABLE III.6

WORST-CASE RISK ASSESSMENT  
CURRENT SITUATION

Daily Dose ( $\mu\text{g}/\text{day}$ ) and Margin of Safety<sup>1</sup>

	Air Pathway		Groundwater Pathway <sup>2</sup>			Surface Water Pathway
	Workers <sup>3</sup>	Off-Site Population	Minerva Well	Sandy Creek	Potential Private Wells <sup>4</sup>	
South Pond	2.8 Adequate to Marginal	0.14 Ample	0.12 Ample	0.01 Redundant	10 Marginal	Small Ample
Swale	0.54 Adequate to Adequate	0.028 Redundant to Ample	0.12 Ample	0.01 Redundant	6 Adequate to Marginal	Medium Adequate
Rubble Pile	0.24 Adequate to Adequate	0.014 Redundant	Small Ample	Small Ample	Small Ample	Medium Adequate
Wax Ditch	-----Insignificant----- Redundant					Small Ample
Fry Property	-----Insignificant----- Redundant					Small Ample

<sup>1</sup>Based on lifetime exposure of which the probability is very small.

<sup>2</sup>Off-site exposure through groundwater would not occur for at least 100 years.

<sup>3</sup>Probability of 40-hours/week exposure is small.

<sup>4</sup>No private wells are currently in place; probability of future wells is uncertain and probably small.



TABLE III.7

MOST PROBABLE-CASE RISK ASSESSMENT  
CURRENT SITUATION

Daily Dose ( $\mu\text{g}/\text{day}$ ) and Margin of Safety<sup>1</sup>

	Air Pathway		Groundwater Pathway <sup>2</sup>			Surface Water Pathway
	Workers <sup>3</sup>	Off-Site Population	Minerva Well	Sandy Creek	Potential Private Wells <sup>4</sup>	
South Pond	0.66 Ample to Adequate	0.14 Ample	<0.01 Redundant	<0.01 Redundant	0.07 Ample	Small Ample
Swale	0.54 Ample to Adequate	0.028 Redundant to Ample	<0.01 Redundant	<0.01 Redundant	0.16 Ample	Medium Adequate
Rubble Pile	0.09 Ample	0.014 Redundant	Small Ample	Small Ample	Small Ample	Medium Adequate
Wax Ditch	-----Insignificant----- Redundant					Small Ample
Fry Property	-----Insignificant----- Redundant					Small Ample

<sup>1</sup>Based on lifetime exposure of which the probability is very small.

<sup>2</sup>Off-site exposure through groundwater would not occur for at least 100 years.

<sup>3</sup>Probability of 40-hours/week exposure is small.

<sup>4</sup>No private wells are currently in place. Probability of future wells is uncertain and probably small

toxicity assessment. Table III.7 depicts probable-case values in a similar manner.

The data presented in Table III.6 indicate that present and future risks posed by PCB contamination at the site are generally insignificant. There is only one scenario leading to potential exposure where the margin of safety is predicted to be less than adequate. If individuals employ small private wells as a source of drinking water in a limited area immediately south of the property, the margin of safety may be marginal. However, this scenario is based on the hypothesis that such wells will be drilled, that a groundwater contaminant plume will develop, and that the plume will move off-site. Such a situation would develop, if at all, only after several hundred years.

A question aside from that of risk concerns the potential for migration of residual PCBs. In this evaluation, we have identified two areas of concern: the potential for groundwater contamination and the potential for contamination of Sandy Creek and the neighboring property from surface runoff. Although the data are not conclusive, we believe there is only a small possibility that residual PCBs will migrate via these transport mechanisms even over an extremely protracted period.

#### IV. REMEDIAL ACTION PLAN

Based on the above risk assessment, residual PCB concentrations at the TRW facility present negligible risks to public health and the environment even if no remedial action was undertaken. The data in Tables III.6 and III.7 indicate, however, that margins of safety vary depending on the source of contamination (e.g., swale, South Pond) and the transport mechanism considered. To emphasize these differences, those scenarios having the least margins of safety have been boxed. Solid boxes represent areas of some concern while dashed boxes represent areas where improvement might prove beneficial. Thus, to further mitigate the potential for PCB migration and increase margins-of-safety, a variety of remedial alternatives were evaluated. Based on this evaluation, the following remedial action plan was developed. Scheduling and monitoring requirements are presented in later sections. Results of an evaluation of the effectiveness of the proposed remedial measures are presented in the final section of this chapter.

##### A. Remedial Action Plan

The areas involved in the cleanup plan are shown in the site layout (Figure IV.1) and encompass three elements:

- Excavation and removal of upper 6-24 inches of surface soil in the swale, rubble pile, Fry property, and wax ditch areas containing the majority of the PCB residuals and replacement with either fresh fill soil or a clay cap depending on the need to reduce infiltration

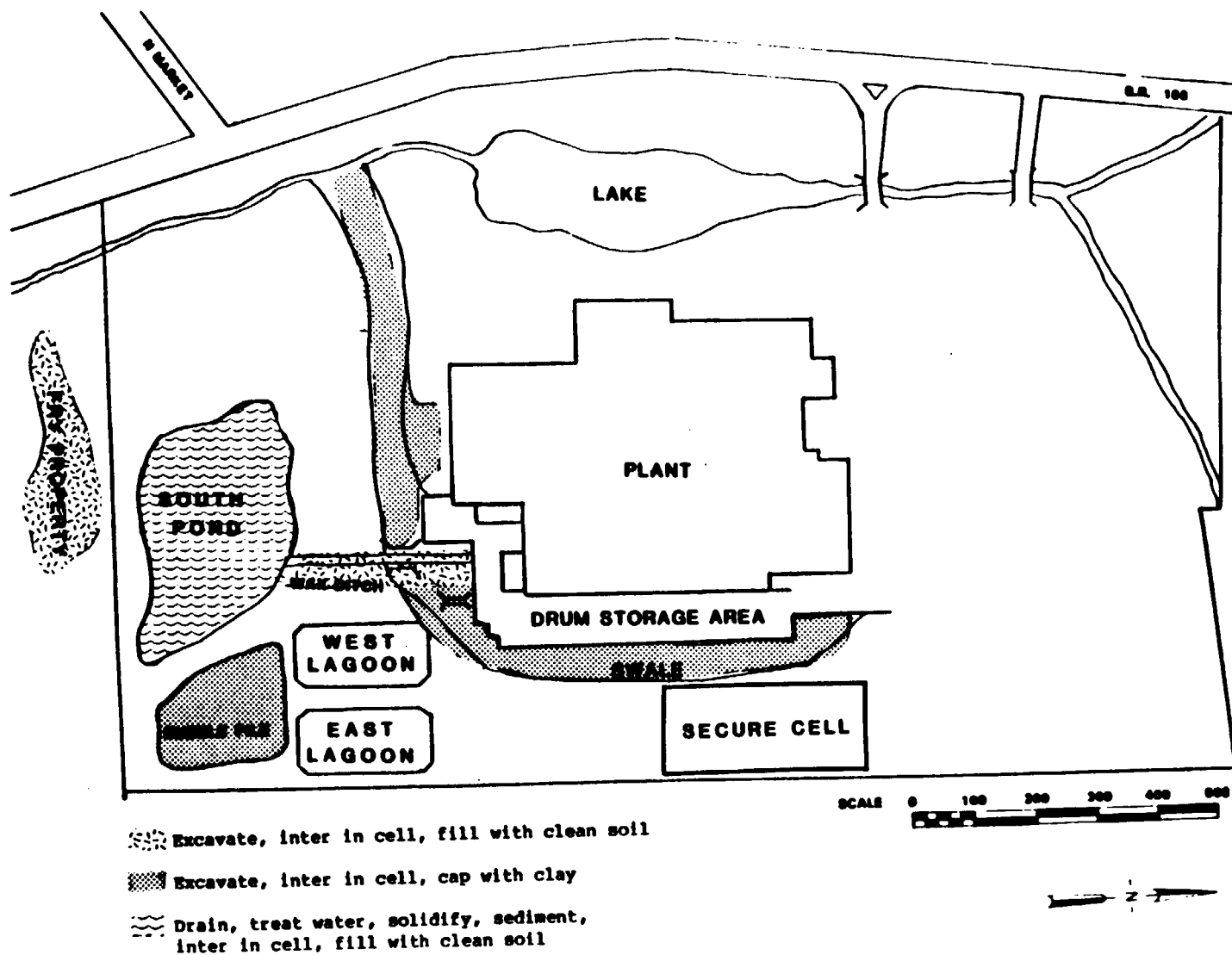


FIGURE IV.1

PROPOSED REMEDIAL ACTIONS AT TRW MINERVA SITE

- Drainage and treatment by carbon adsorption of the South Pond water with subsequent solidification and removal of the pond sediment
- Construction of a secure cell on the plant property for interment of the excavated PCB-containing materials and solidified pond sediments

A summary of the objectives of the remedial action plan and proposed actions for mitigating the problems cited is presented in Table IV.1. Details of the specific remedial actions proposed for each of the areas on the site that contain residual PCBs are presented below. The design and proposed location for the secure interment cell are also presented. Area and volume estimates for the clean-up areas are shown in Table IV.2.

#### 1. Remedial Measures for Areas Containing Residual PCBs

A summary of remediation plans for each of the areas of the site exhibiting residual PCB concentrations is presented in this section. Clean-up objectives are stated under each area heading followed by a list of remedial actions designed to meet the objectives stated.

##### a. South Pond

Objective--to increase the margin of safety for potential private, off-site wells and on-site worker exposure

##### Remedial Plan

- Remove, treat and discharge the pond water to the West Lake drainage system by NPDES permit (time estimate--6-8 weeks)
  - Prepare a discharge drainage piping system (200 yards) to the West Lake stream
  - Treat pond water (2.25 million gallons) by pumping through mobile carbon adsorption treatment system and discharge into drainage system; spent carbon cartridge disposed on-site (25 yd<sup>3</sup>) in secure cell.

TABLE IV.1

## SUMMARY OF REMEDIAL ACTION PLAN--MIGRATION PATHWAY MITIGATION

Area	Migration Pathway	Mitigation Approach
Swale and Rubble Pile	Surface water via erosion and rainwater runoff	Surface sealing with 1.5-2 foot clay cap to retard erosion, surface water infiltration, and PCB volatilization
	Groundwater via rainfall infiltration and percolation	
	Air via volatilization from from surface	
South Pond	Surface water via dike failure	Elimination of pond by treating and discharging pond water, solidifying pond sediments, and removing to on-site secure cell
	Groundwater via percolation of pond water through pond pond liner	
	Air via volatilization from pond surface	
Wax Ditch	Surface water via erosion of ditch sediments	Elimination of ditch by removing waxy materials and sediments, interring in on-site secure cell, and backfilling ditch with clean soil
Fry Property	Surface water via erosion or washout of surface contaminated soils	Elimination of shallow surface contaminated areas by removing to on-site secure cell, and backfilling with clean soil

TABLE IV.2  
ESTIMATES FOR VOLUMES AND SURFACE AREAS  
OF MATERIALS TO BE MOVED

Area	Excavation depth (ft)	Surface Area		Volume	
		ft <sup>2</sup>	yd <sup>2</sup>	ft <sup>3</sup>	yd <sup>3</sup>
Swale					
Section A	1.5	25,250	2,806	37,875	1,403
Section B	1.5	39,375	4,375	59,063	2,188
Section C	2	26,407	2,934	52,814	1,956
		91,032	10,115	149,752	5,547
			Contingency <sup>b</sup>	748	
					6,295
Wax Ditch	2.5	3,000	333	7,500	280
Pond Sediments	1	77,500	8,611	77,500	5,740 <sup>c</sup>
Rubble Pile	1	77,500	8,611	60,000	2,222
Fry Property	0.5	25,000	2,778	12,600	463
Total Estimated yd <sup>2</sup>			30,448 yd <sup>2</sup>	15,000 yd <sup>3c</sup>	

<sup>a</sup>Area and volume estimates vary from Table II.1 to accommodate excavation practices and requirements of the remedial plan

<sup>b</sup>748 yd<sup>3</sup> contingency added for remaining A area and wider removal zones

<sup>c</sup>Includes 2,870 yd<sup>3</sup> with 1:1 kiln dust additions

- Solidify the sediment in the pond, remove, and inter the solidified sediment in a secure on-site cell (time estimate--2 weeks)
  - Following drainage and treatment of pond water, solidify sediment with kiln dust (1:1 ratio), and remove solidified material, together with approximately 1-foot depth of pond liner (6,000 yd<sup>3</sup>)
  - Install sediment control barrier (straw/fencing) to contain erosion sediment during construction period
  - Place the excavated material in the secure on-site cell
- Fill pond area with berm material (and borrowed material if necessary) and grade to provide channeled surface run-off toward West Lake drainage stream and seed with natural grasses
  - Backfill requirements--1,000 yd<sup>3</sup>
  - Seed area--8,611 yd<sup>2</sup>

b. Swale

Objective--to increase margin of safety for potential private wells, surface run-off and on-site worker exposure

Remedial Plan

- Remove surface contaminated soil areas in Sections A and B and in corner Section C; inter this material in secure on-site cell.
  - Establish sediment control system (gravel/riprap--10 yds<sup>3</sup>)
  - Excavate 10,115 yd<sup>2</sup> area (Sections A & B--7,185 yd<sup>2</sup> at 1.5 ft depth; Section C (corner)--2,930 yd<sup>2</sup> at 2-ft depth)
  - Remove and place in on-site secure cell (6,300 yd<sup>3</sup>)
- Grade and fill and cap with compacted clay to provide a drainage channel sufficient to rapidly drain run-off, minimizing standing water and infiltration, add 6-12 inches of top soil, and seed with natural grasses
  - Grade, place compacted clay cap to original level (1.5 ft, Sections A & B; 2 ft, Section C)



- Clay requirements--6,500 yd<sup>3</sup>
- Top soil requirements--5,000 yd<sup>3</sup>
- Seed area--10,111 yd<sup>2</sup>

c. Rubble Pile

Objective--to increase margin of safety for surface water run-off and on-site worker exposure

Remedial Plan

- Prepare sediment control barrier and rainfall diversion ditch along north fence line
- Remove contaminated debris and soil and inter in secure on-site cell
  - Excavate large debris and top 6-12 inches of soil and debris
  - Remove and place in on-site secure cell--2,225 yd<sup>3</sup>
- Grade to achieve 2% minimum grade, cap with 1-1.5 feet of clay, add 6-12 inches of top soil, and seed with natural grasses
  - Clay requirement--3,350 yd<sup>3</sup>
  - Top soil requirement--1,675 yd<sup>3</sup>
  - Seed area--8,611 yd<sup>2</sup>

d. Wax Ditch

Objective--to remove all surface materials containing highest PCB concentrations to avoid any potential wash-out or other transport of contaminated soil to surface waters

Remedial Plan

- Remove wax deposits and underlying soil and inter in secure on-site cell
  - Excavate ditch materials plus an additional 1 foot of soil (2.5 foot total)
  - Remove and place in secure cell--230 yd<sup>3</sup>

- Backfill to surface grade level with clean borrowed soil, grade, compact, and seed with natural grasses
  - Backfill soil requirements--1,000 yd<sup>3</sup>
  - Seed area--333 yd<sup>2</sup>

e. Fry Property

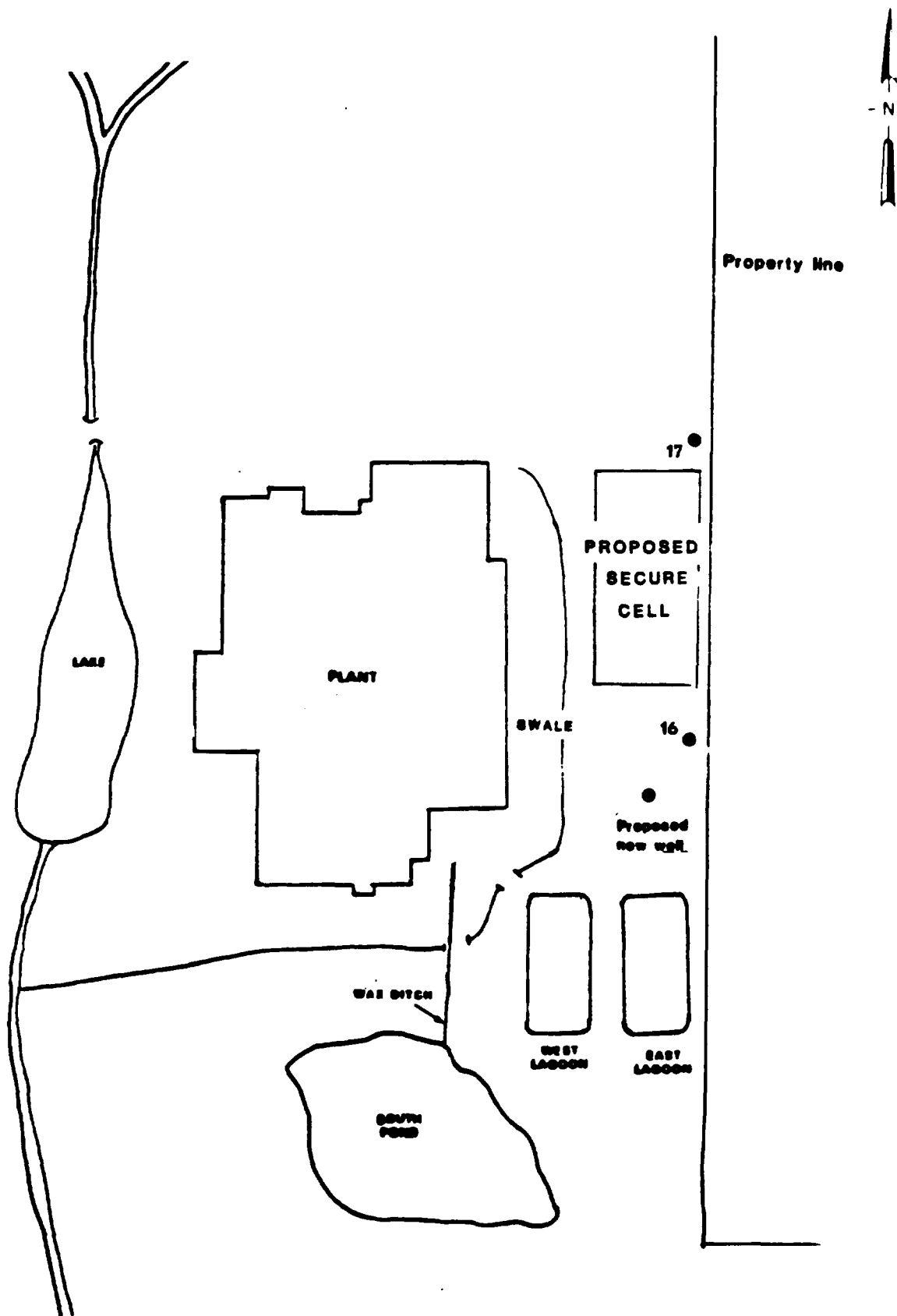
Objective--to remove the most contaminated surface materials to avoid any washout or other transport of contaminated soil to surface waters

Remedial Plan

- Remove soil in areas having high concentrations and inter in secure on-site cell
  - Remove brush--3,000 yd<sup>2</sup>
  - Excavate contaminated soil at 6-inch depth--25,000 ft<sup>2</sup>
  - Remove and place in secure cell--500 yd<sup>3</sup>
- Fill and grade with borrowed soil from the site and seed with natural grasses
  - Backfill requirements--500 yd<sup>3</sup>
  - Seed area--2,800 yd<sup>2</sup>

2. Construction of a Secure Interment Cell

The construction of an on-site secure cell was recommended in order to inter PCB-containing soil materials excavated as a part of the above remedial action plan. A fairly flat area immediately west of the plant between monitoring wells 17 and 16/15 was selected for the construction of the secure cell. The proposed location of the secure cell and the wells for monitoring the cell are shown in Figure IV.2. One additional monitoring well is proposed to be installed as indicated.



**FIGURE IV.2**

**LOCATION OF GROUNDWATER SAMPLING WELLS  
AND PROPOSED SECURE CELL**

The secure cell has been designed to meet the requirements specified in the regulations for PCB-containing soil materials landfilled (40CFR761.71).

a. Construction

The cell will be constructed with a 3-foot clay liner, a leachate collection system, and a compact clay perimeter berm. All side slopes will be at a grade of 3:1 in order to minimize the potential for sloughing and erosion. The cell will be designed to ensure that the bottom of the cell liner is at least 3 feet above the highest observed groundwater table.

After the excavated materials have been placed in the cell and are graded and compacted, a compacted 2-foot clay cap and 6-12 inches of top soil will be installed. Both the clay cap and the surrounding area will be seeded with natural grasses to provide slope stabilization and erosion control. Specific details for the cell design liner construction and leachate collection system are shown in Figures IV.3 and IV.4. Estimates of the clay requirements for cell liner and cap construction can be found in Table IV.3. Proposed cell dimensions are as follows:

- Finished cell before filling
  - Surface area: berm interior 125x350 ft; 43,750 ft<sup>2</sup>, 4,861 yd<sup>2</sup>
  - Depth: varies from 4 to 8.5 ft from top of berm
  - Height above ground level: 6 ft
- Completed secure cell after filling
  - Dimensions: 125x350x13 ft (average)

**TABLE IV.3**  
**ESTIMATE OF SECURE CELL CLAY REQUIREMENTS**

Area	Dimensions	Volume	
	(feet)	ft <sup>3</sup>	yd <sup>3</sup>
Bottom	326x101x3	98,778	
Sides (4)	2(350x12x3) + 2(125x12x3)	34,200	
Berm (4 sides)	2(6x18x350) + 2(6x6x350) + 2(6x18x150) + 2(6x6x125)	136,800	
		269,778	9,992
Cap	(350x126x2) + (350x13x2) + 2(126x24x2)	100,576	3,725
Basic Clay Requirements		339,076	13,717

- Surface area: interior 398x173 ft; 68,854 ft<sup>2</sup>, 7,650 yd<sup>2</sup>
- Depth: varies from 8 to 18 ft
- Containment volume: 15,000 yd<sup>3</sup>
- Height above ground level: 16 ft

A summary of the amount of PCB-containing material expected to be placed within the secure cell (approximately 15,000 yd<sup>3</sup>) is shown in Table IV.4. The materials will be placed in the cell, graded, and compacted in order to minimize future subsidence. Haul roads from the site work areas will be kept to a minimum, and the roads will be watered for dust control during operation.

Surface run-off from surrounding areas will be prevented from draining into the secure cell by the perimeter compacted clay berm. Direct precipitation into the secure cell during operation will be collected by the leachate collection system, which will be operated as described above.

All personnel operating in or around the secure cell and on the site will be required to wear disposable coveralls, boots and gloves, hard hats, and dust masks. Disposable coveralls, boots, and gloves will be discarded and placed in the secure cell. Hard hats and dust masks will not be allowed to be taken off-site. At the end of the remedial action, they will be decontaminated or discarded by being placed in the secure cell. The contractor selected to perform the remedial action will operate the secure cell. That contractor will be required to maintain state-of-the-art worker health and safety procedures for both operations.

TABLE IV.4

QUANTITY AND CHARACTERIZATION OF MATERIALS  
TO BE PLACED IN THE SECURE CELL

Site Area/Material	Volume of Material to be Landfilled (yd <sup>3</sup> )	Concentration of PCBs in Material (ppm)
Swale: Soil	6300	Average: 350 Range: <1-1,000
South Pond: Solidified Sediment	5,740 <sup>a</sup>	Average: 300 <sup>b</sup> Range: <1-2,000
Wax Ditch: Wax Residues	280	Average: 4,000 Range: 2,000-5,000
Rubble Pile: Soil/Debris	2,230	Average: 100 Range: <1-1,000
Fry Property: Soil	470	Average: <10 Range: <1-33,000
Total	15,020	

<sup>a</sup>Includes volume of cement dust added for solidification

<sup>b</sup>Not including the cement dust

Unauthorized entry to the secure cell during operation will be controlled by a fence along the back and sides of the property. Unauthorized access after closure will be prevented by a 6-foot-high chain-link fence installed around the secure cell.

b. Chronology for constructing the secure cell (time estimate 3-4 weeks)

Construction of the on-site internment all for permanent storage of soils and sediments containing residual levels of PCBs will be performed in the following phases:

Cell Excavation

- Excavate cell floor to grade plan

Berm and Liner Construction

- Build 3-ft clay floor in 6-inch lifts and compact to 95% standard Proctor density
- Build similar 3-ft clay side walls (using 3:1 slope) and 6-ft berm around perimeter (to contain residuals and prevent surface water intrusion)
  - Clay requirements--10,000 yd<sup>3</sup>

Leachate Collection System Installation

- Install 1-ft gravel leachate collection field on floor of cell (1x150x350 ft)
  - Install leachate collection trench and 4-inch perforated PVC pipe; install 4-ft manhole/sump standpipe in center of cell floor
  - Gravel requirements--1x126x326 ft + 1x1x326 ft, 1,533 yd<sup>3</sup> (1-2 inch crushed stone)
  - Construction fabric cloth--15 rolls (400x10 ft)
  - Perforated PVC pipe--4 in diameter x 330 ft (schedule 80)



- To install sump with manhole/standpipe with
  - 6 ft diameter precast reinforced concrete pad
  - 4' diameter x 24' precast reinforced concrete standpipe perforated with 1" inch holes (<25% of area) on lower 3'

#### Cell Filling

- Construction of haul roads and fill ramp within secure cell
- Placement and compaction of PCB-containing soils and materials in maximum of 3-ft lifts
- After completion of remedial operation, the haul roads will be scraped (3 inches) and the soil will be removed and placed in the secure cell
- Grade for final cover

#### Cell Closure and Cap Installation

- After the cell is filled with the excavated soil materials, install a minimum 2-ft clay cap and topsoil cover 6-12 inch according to the construction plan and seed with natural grasses
  - Clay requirements--2,725 yd<sup>3</sup>

Grade for surface run-off drainage--5% slopes on top surface, 3:1 slope on side surfaces
  - Topsoil requirements--1,275 yd<sup>3</sup>
  - Seed area--7,650 yd<sup>3</sup>

#### B. Schedule for Remedial Actions--Critical Path

The critical path timetable required to complete the proposed site remedial activities is shown in Table IV.5. This timetable requires that intensive site work be undertaken for 18-20 weeks (barring weather delays) after plan approval and contractor selection. Prompt approval of the proposed remedial action plan and secure cell design is important to complete the project within the 1983 construction season.

TABLE IV.5  
CLEAN-UP ACTIVITIES  
CRITICAL PATH

Total Elapsed Time 18-20 Weeks Without Weather Delays

<u>Phase I</u>		(a) Mobilization--2 weeks
10 weeks		Sediment control/diking; preparation of South Pond drainage area
		(b) Clean-up activities--8 weeks
		South Pond drainage/treatment water/carbon adsorption
		Secure cell preparation
<u>Phase II</u>		(a) Excavation site areas/fill replacement--3 weeks
7 weeks		Swale 2 weeks
		Rubble pile 1 week
		Fry property 2 weeks
		Wax ditch 1 week
		(b) Sediment treatment/excavation--2 weeks
		South Pond
		(c) Clay cap installation--2-3 weeks
		Swale 1 week
		Rubble pile 1 week
		(d) Seeding/stabilization--1 week
<u>Phase III</u>		(a) Equipment decontamination/demobilization--2 weeks
2 weeks		Removal of sediment control measures
intermittent		(b) Continued well monitoring/stream sediments
monitoring		During site work--monthly samples
for 20 years		After remedial work completed--semiannual

### C. Site Monitoring Program

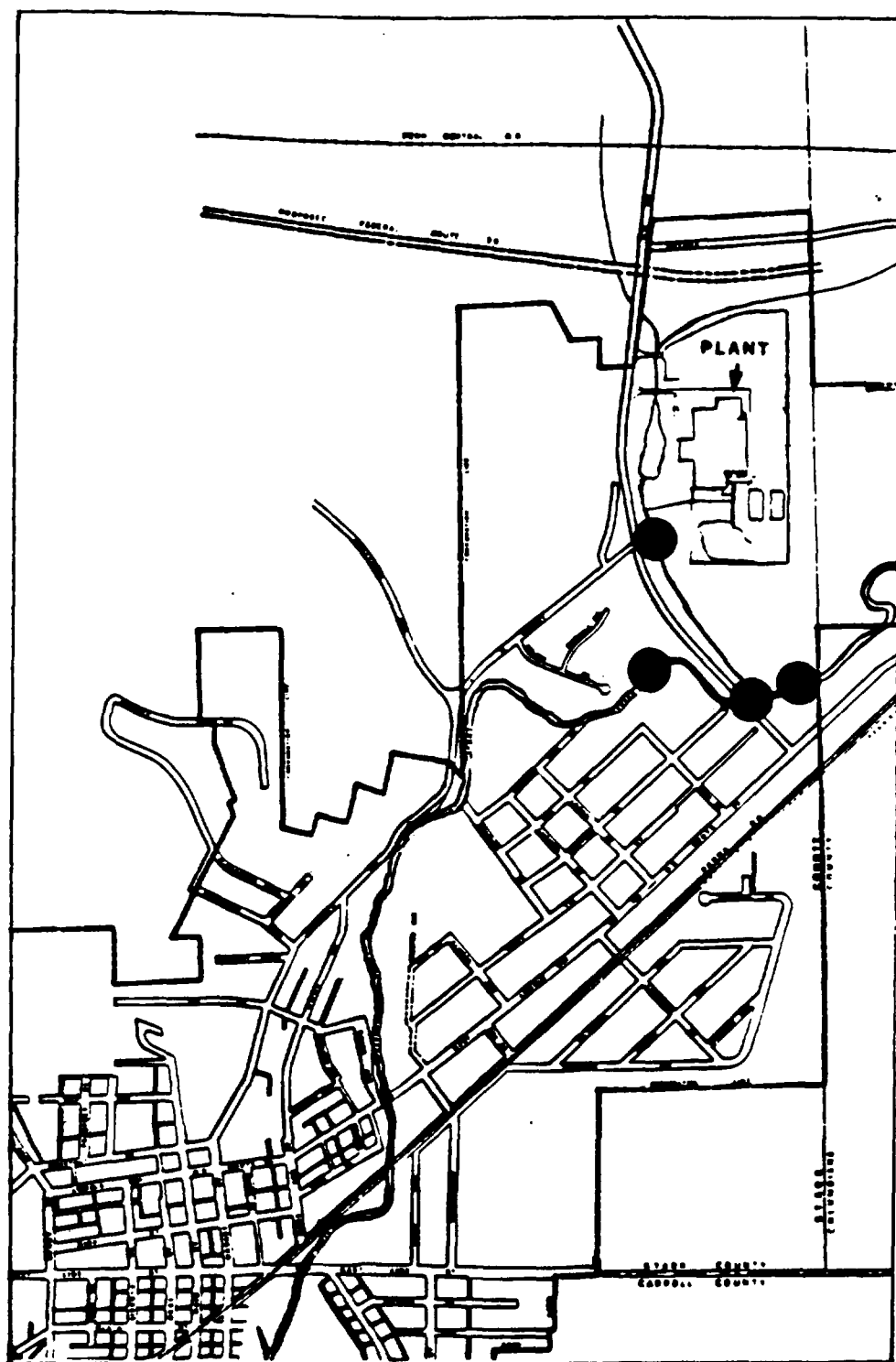
During remedial work at the site and construction of the secure cell and for 20 years thereafter, various monitoring activities are proposed. The following monitoring will be performed (locations of surface water monitoring points are shown on Figure IV.5):

#### During Remedial Work

- Monthly monitoring of six selected downgradient wells on the Fry property and one upgradient well (well 17). In addition, two wells downgradient from the secure cell (one of which will be a new well) will be monitored monthly
- Monthly monitoring of surface water at two points on the stream emanating from West Lake and two points in Sandy Creek
- Monthly monitoring of sediment runoff at the mouth of the swale and at the junction of the stream emanating from West Lake and Sandy Creek
- Monthly monitoring for leachate of secure cell sump. At a depth of 30 centimeters (1.5 feet) the leachate will be removed and sent offsite to an approved facility for treatment

#### After Completion of Remedial Work

- Monthly monitoring of secure cell sump for leachate for a 3-year period following cell closure, then semi-annually for 17 years. At a depth of 30 centimeters (1.5 feet) the leachate will be removed and sent off-site to an approved facility for treatment
- Continued semiannual monitoring of three selected groundwater wells on the Fry property and the three wells associated with the secure cell
- Continued semiannual monitoring of sediments at the two sediment monitoring stations established



● Sampling point

Note: All points will be sampled monthly during operation of the landfill. The westernmost points will be sampled semiannually after closure of the landfill.

**FIGURE IV.5**  
**SURFACE WATER SAMPLING POINTS**

The analysis to be performed on the well, surface water sediments and leachate samples collected during the remedial work period and after cell closure will be as follows:

#### Water Analysis

All groundwater and surface water samples will be analyzed for PCBs, pH, specific conductance, and chlorinated organics (as measured by total organic halogens) pursuant to methods specified in 40 CFR 761.71. Results of the water analyses will be maintained for 20 years after the closure of the landfill.

#### Sediment Analysis

Sediment samples collected will be analyzed for PCB content.

#### D. Remedial Action Risk Assessment

A second risk assessment was performed considering site modifications that would result from the above remedial measures. Numbers were derived as indicated in the risk assessment. PCB transport via volatilization and sediment runoff from the secure cell will be insignificant because of the clay barrier. Percolation was modeled assuming a saturated zone up to 1-foot thick might develop within the cell before it would be detected by the leachate collection system. Percolation driven by this 1-foot head would then have to traverse 3 feet of  $1 \times 10^{-7}$  cm/second clay. The rest of the calculations parallel those for other regions onsite.

Results of this assessment are summarized in Table IV.6. The data displayed in this table indicate that the prescribed

TABLE IV.6

WORST-CASE RISK ASSESSMENT  
AFTER REMEDIAL ACTION

Daily Dose ( $\mu\text{g}/\text{day}$ ) and Margin of Safety<sup>1</sup>

	Air Pathway		Groundwater Pathway <sup>2</sup>			Surface Water Pathway
	Workers <sup>3</sup>	Off-Site Population	Minerva Well	Sandy Creek	Potential Private Wells <sup>4</sup>	
South Pond	-----Insignificant----- Redundant					
Swale	<0.01 Redundant	<0.01 Redundant	<0.01 Redundant	<0.01 Redundant	0.07 Redundant to Ample	Insignificant Redundant
103 Rubble Pile	-----Insignificant----- Redundant					
Wax Ditch	-----Insignificant----- Redundant					
Fry Property	-----Insignificant----- Redundant					
Secure Cell	-----Insignificant----- Redundant					

<sup>1</sup>Based on lifetime exposure of which the probability is very small.

<sup>2</sup>Off-site exposure through groundwater would not occur for at least 100 years.

<sup>3</sup>Probability of 40-hours/week exposure is small.

<sup>4</sup>No private wells are currently in place. Probability of future wells is uncertain and probably small.

measures would be extremely effective. Covering and capping all potential sources on the site effectively eliminate volatilization and sediment runoff as potential exposure pathways. A combination of removing the majority of residual PCBs present and grading and capping (to minimize percolation) effectively eliminates contributions from these sources to groundwater contamination.

## REFERENCES

- ALLEN, J., BARSOTTI, D., LAMBRECHT, L., and VAN MILLER, J. 1979. Reproductive effects of halogenated aromatic hydrocarbons on nonhuman primates. *Ann. NY Acad. Sci.* 320:419-425
- ALLEN, J., BARSOTTI, D., and CARSTENS, L., 1980. Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. *J. Toxicol. Environ. Health* 6:55-66
- BAHN, A.K., ROSENWAIKE, I., HERRMANN, N., GROVER, P., STELLMAN, J., and O'LEARY, K. 1976. Melanoma after exposure to PCB's. *N. Engl. J. Med.* 295:450
- BAKER, E.L., Jr., LANDRIGAN, P.J., GLUECK, C.J., ZACK, M.M., Jr., LIDDLE, J.A., BURSE, V.W., HOUSWORTH, W.J., and NEEDHAM, L.L. 1980. Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. *Am. J. Epidemiol.* 112:553-563
- BERCOVICI, B., WASSERMANN, M., WASSERMANN, D., CUCOS, S., and RON, M. 1980. Missed abortions and some organochlorine compounds: Organochlorine insecticides (OCI) and polychlorinated biphenyls (PCBs). *Acta Med. Leg. Soc.* 30:177-185
- BERTAZZI, P.A., ZOCCHETTI, C., GUERCILENA, S., FOGLIA, M.S., PESATORI, A., and RIBOLDI, L. 1981. Mortality study of male and female workers exposed to PCBs. Paper presented at the International Symposium on Prevention of Occupational Cancer, April 21-24, 1981, Helsinki, Finland
- BOWES, G.W., MULVIHILL, M.J., SIMONEIT, B.R.T., BURLINGAME, A.L., and RISEBROUGH, R.W. 1975. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. *Nature* 256:305-307
- BOWMAN, R., HEIRONIMUS, M., and ALLEN, J. 1978. Correlations of PCB body burden with behavioral toxicology in monkeys. *Pharmacol. Biochem. Behav.* 9:49-56
- BOWMAN, R., HEIRONIMUS, M., and BARSOTTI, D. 1981. Locomotor hyperactivity in PCB-exposed Rhesus monkeys. *Neurotoxicology* 2:251-268
- BROWN, D.P., and JONES, M. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. *Arch. Environ. Health* 36:120-129



- CHANG, K.J., CHENG, J.S., HUANG, P.C., and TUNG, T.C. 1980. Study of patients with PCB poisoning. J. Formosan Med. Assoc. 79:304-313
- CRUMP, K.S. and MASTERMAN, E. 1979. Assessment of Carcinogenic Risks from Exposure to PCBs. Unpublished Report to U.S. Environmental Protection Agency.
- Di TORO, M.D., and HORZEMPA, L.M. 1982. Reversible and Resistant Components of PCB Adsorption-Desorption: Isotherms. Environ. Sci. Technol. 16:594-602
- EARL, F., COUVILLION, J. and Van LOON, E. 1974. The reproductive effects of PCB 1254 in beagle dogs and miniature swine. Toxicol. Appl. Pharmacol. 29:104 (Abstract)
- FISCHBEIN, A., WOLFF, M.S., LILIS, R., THORNTON, J., and SELIKOFF, I.J. 1979. Clinical findings among PCB-exposed capacitor manufacturing workers. Ann. NY Acad. Sci. 320:703-715
- HARTLEY, G.S. 1969. Evaporation of pesticides. In Advances in Chemistry. Series 86. Chapter 11. American Chemical Society.
- HAYABACHI, H., YOSHIMURA, T., and KURATSUNE, M. 1979. Consumption of toxic rice oil by 'Yusho' patients and relation to the clinical response and latent period. Food Cosmet. Toxicol. 17:455-461
- HUMPHREY, H.E.B. 1977. Evaluation of Changes of the Level of Polychlorinated Biphenyls (PCB) in Human Tissue. Final Report on FDA Contract 223-73-2209
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 18: Polychlorinated Biphenyls and Polybrominated Biphenyls. World Health Organization, Lyon, France
- KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J., and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. JNCI 55:1453-1456
- KREISS, K., ZACK, M.M., KIMBROUGH, R.D., NEEDHAM, L.L., SMREK, A.L., and JONES, B.T. 1981. Association of blood pressure and polychlorinated biphenyl levels. JAMA 245:2505-2509
- KURATSUNE, M., YOSHIMURA, T., MATSUZAKA, J., and YAMAGUCHI, A. 1972. Epidemiological study on Yusho, a poisoning caused by ingestion of rice oil contaminated with a commercial brand of polychlorinated biphenyls. Environ. Health Perspect. 1:119-128

- KURATSUNE, M., MASUDA, Y., and NAGAYAMA, J. 1976. Some of the recent findings concerning Yusho. Proceedings of the National Conference on Polychlorinated Biphenyls. Pp. 15-37
- LUCIER, G., DAVIS, G., and McLACHLAN, J. 1978. Transplacental toxicology of the polychlorinated and polybrominated biphenyls. Dept. of Energy Symposia Series Vol. 47: Toxicology of Energy-Related Pollutants. Pp. 188-203
- MACKAY, D., and LEINONEN, P.J. 1975. Rate of evaporation of low-solubility contaminants from water bodies to atmosphere. Environ. Sci. Tech. 9:1178-1180
- McNULTY, W.P., POMERANTZ, I., and FARRELL, T. 1981. Chronic toxicity of 2,3,7,8-tetrachlorodibenzofuran for Rhesus macaques. Food Cosmet. Toxicol. 19:57-65
- MORALES, N.M., and MATTHEWS, H.B. 1979. In vivo binding of 2,3,6,2',3',6'-hexachlorobiphenyl and 2,4,5,2',4',5'-hexachlorobiphenyl to mouse liver macromolecules. Chem. Biol. Interact. 27:99-110
- NATIONAL ACADEMY OF SCIENCES (NAS). 1977. Drinking Water and Health. NAS, Washington, D.C.
- NATIONAL ACADEMY OF SCIENCES (NAS) 1979. Polychlorinated Biphenyls. NAS, Washington, D.C.
- NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. NCI Carcinogenesis Technical Report No. 38
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Health Hazard Evaluation Determination of Westinghouse Electric Corp., Bloomington, Indiana. Report No. 76-52-386. NTIS PB-273 735. Cincinnati, Ohio
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Polychlorinated Biphenyls (PCBs). DHEW Publication No. (NIOSH) 77-225
- NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION (NOAA). 1981. Local and Climatological Data: Annual Summary with Comparative Data. Cleveland Ohio. National Climatic Center, Federal Building, Asheville, North Carolina, 28801
- NATIONAL RESEARCH COUNCIL OF CANADA. 1978. Polychlorinated Biphenyls: Biological Criteria for an Assessment of Their Effects on Environmental Quality. NRCC No. 16077

- NISHIZUMI, M. 1976. Enhancement of diethylnitrosamine hepatocarcinogenesis in rats by exposure to polychlorinated biphenyls or phenobarbital. *Cancer Lett.* 2:11-16
- NISHIZUMI, M. 1979. Effect of phenobarbital, dichlorodiphenyl-trichloroethane and polychlorinated biphenyls on diethylnitrosamine-induced hepatocarcinogenesis. *Gann* 70:835-837
- PRESTON, B.D., VAN MILLER, J.P., MOORE, R.W., and ALLEN, J.R. 1981. Promoting effects of polychlorinated biphenyls (Aroclor 1254) and polychlorinated dibenzofuran-free Aroclor 1254 on diethylnitrosamine-induced tumorigenesis in the rat. *JNCI* 66:509-515
- THIBODEAUX, L.J. 1979. *Chemodynamics: Environmental Movement of Chemicals in Air, Water, and Soil.* John Wiley & Sons, New York
- TOFFLEMIRE, T.J., and SHEN, T.T. 1979. Volatization of PCB from sediment and water: Experimental and field data. *Proceedings of the Mid-Atlantic Waste Conference.* Pp. 100-109
- TURNER, D.B. 1970. *Workbook of Atmospheric Dispersion Estimates.* USDHEW, Public Health Service, Environmental Health Service Publication No. 999-AP-26, National Air Pollution Control Administration, Cincinnati, Ohio
- UNGER, M., AND OLSEN, J. 1980. Organochlorine compounds in the adipose tissue of deceased people with and without cancer. *Environ. Res.* 23:257-263
- U.S. DEPARTMENT OF AGRICULTURE (USDA). 1971. *Soil Survey: Stark County, Ohio.* Superintendent of Documents. USGPO, Washington, D.C., 20402. October 1971
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA) 1980a. *Attenuation of Water Soluble Polychlorinated Biphenyls by Earth Materials.* EPA-600/2-80-027, May 1980
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA) 1980b. *Ambient Water Quality Criteria for Polychlorinated Biphenyls.* EPA-440/5-80-068 October 1980
- WARSHAW, R., FISCHBEIN, A., THORNTON, J., MILLER, A., and SELIKOFF, I.J. 1979. Decrease in vital capacity in PCB-exposed workers in a capacitor manufacturing facility. *Ann. NY Acad. Sci.* 320:277-283
- WASSERMANN, M., RON, M., BERCOVICI, B., WASSERMANN, D., CUCOS, S., and PINES, A. 1982. Premature delivery and organochlorine compounds: Polychlorinated biphenyls and some organochlorine insecticides. *Environ. Res.* 28:106-112

WORLD HEALTH ORGANIZATION (WHO). 1978. Environmental Health Criteria for Polychlorinated Biphenyls and Polychlorinated Terphenyls. WHO, Geneva, Switzerland

WYNDHAM, C., DEVENISH, J., and SAFE, S. 1976. The in vitro metabolism, macromolecular binding and bacterial mutagenicity of 4-chlorobiphenyl, a model PCB substrate. Res. Commun. Chem. Pathol. Pharmacol. 15:563-570